THE JOURNAL

OF THE

OKLAHOMA STATE MEDICAL ASSOCIATION

VOLUME XXVI MUSKOGEE, OKLAHOMA, AUGUST, 1933. NUMBER 8

A SYMPOSIUM ON DIABETES

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Presented before Section on General Medicine, Forty-first Annual Session, Oklahoma State Medical Association, Oklahoma City, May 16, 1933

DIAGNOSIS OF DIABETES MELLITUS —SIGNIFICANCE OF GLYCOSURIA

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OUTLINE

Diagnosis of Diabetes Mellitus; Significance of Glycosuria.

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INTRODUCTION

The diagnosis of diabetes is not, as a rule, attended with any unusual difficulty. However, certain factors are worthy of consideration in the establishment of a diagnosis, and it is the purpose of this paper to consider some of the more important ones. It is commonly accepted that the finding of sugar in the urine is diagnostic of diabetes. This is true in a vast majority of cases. Within the last one or two decades the increasing number of insurance examinations and the routine industrial examinations of employees have brought about the discovery of a considerable number of cases, which would have otherwise gone on unrecognized. This perhaps explains the increasing incidence of diabetes as shown by statistics[1]. Whether the present day mode of living, the modern diet and increasing tendency toward a sedentary life, are responsible for the growing incidence of this disease, has not as yet been shown. It would perhaps be more logical to assume that better diagnostic methods, a better education of the laity, and the more frequent examination of individuals, are responsible for the apparent increase in the number of diabetics under treatment today. In view of the gravity of diabetes, the urgent need for treatment, and the present day better methods of management, it behooves the medical profession to acquaint itself with those facts which contribute to better methods of diagnosis. Furthermore, an adequate knowledge of glycosuria, whether it be of diabetic origin or due to other causes, is most essential to the better practice of medicine. The finding of sugar in the urine is not universally diagnostic of diabetes, but every individual in the writer's opinion, who reveals such condition is a potential diabetic until proven otherwise. This may appear to be a broad statement, but if we analyze carefully, the causes of glycosuria, its significance can

be readily appreciated. There is too great a tendency to pass judgment on a case following a hastily or carelessly examined single specimen of urine. The patient examined today who shows a trace of sugar might tomorrow, if properly examined, show a truly diabetic condition. Such a case deserves the examination of not less than three twenty-four hour specimens, and a careful history and clinical examination. The blood sugar may be determined but is not in all cases possible due to lack of facilities and it is not always necessary.

The urinalysis in diabetic diagnosis may be said to be analagous to the Wassermann reaction in the diagnosis of syphilis. However, the actual diagnosis, the clinical findings and other laboratory facilities are available. If such procedures are carried out true diabetes will never be confused with non-diabetic causes of glycosuria[2]. Neither will the true diabetic be overlooked, only to be later discovered in coma, thus jeopardizing his life when his condition might have been recognized and a satisfactory and safe regime of treatment been outlined for him. The writer has within the past two years observed two such deplorable events.

ETIOLOGY

The etiology of diabetes is as yet obscure. One cannot, however, disregard the predisposing factors. A careful history of each case will not infrequently prove quite important not only from the standpoint of diagnosis but will prove valuable from a prognostic point of view. Age is important; the great percentage of cases occur in the fifth and sixth decades of life[3]. Less than five per cent of cases occur during the first decade[3]. Juvenile diabetes is, in the experience of the writer the most malignant form. Not infrequently I have seen it occur at puberty in a very severe form. Statistics indicate that males are more succeptible to the disease than females. The high incidence of the disease in the Jewish race is a well established fact. The Hindus in India are peculiarly susceptible[3]. Heredity is unquestionably of vital importance. Every case should have a careful family history taken. Von Noorden and Pleasants have shown conclusively that there is a definite familial tendency to diabetes mellitus[4].

Diet as a predisposing factor is perhaps exaggerated. However, the writer has found, particularly diabetes in children, to occur in those who have been excessive consumers of sugar, such as candy, fruits, etc. Obesity is without question a factor, but it too is over emphasized[4]. Nervous strain, syphilis, arteriosclerosis, pregnancy, etc., are worthy of mention but not elaboration.

Personally, I feel that acute infections have not received the consideration they merit as a causative factor. During the past three years, I firmly believe that I have observed three severe cases of diabetes to develop following an attack of so-called "flu." We have frequently seen a severe case of acute nephritis follow an acute tonsillitis or scarlitina; or an endocarditis follow an acute follicular tonsillitis, or acute rheumatism. Is it not logical to infer that a fulminating attack of diabetes might follow an acute infection, due to the toxic affect of the disease upon the pancreas. I am also of the opinion that there is a wide difference between the cause of diabetes in the young and that in the older individuals. Diabetes in advanced life seems to be the end result of degenerative changes; in the young the result of the action of a toxin.

There has been many theories advanced to explain the cause of diabetes. None have proven satisfactory, and they have one by one gradually been abandoned. At present it must be admitted that the direct exciting cause has not been discovered. As regards disease of the pancreas, there is no consistent finding, to which the etiology can be attributed.

PATHOLOGY

In 1928 the writer received the necropsy reports on all fatal cases of diabetes mellitus which had come to postmortem examination at the Peter Bent Brigham Hospital in Boston, over a period of fifteen years[5]. The total number of cases was forty-seven. Gross pathology of the pancreas was not remarkable. The histopathology revealed the following findings:

Four revealed hyaline degeneration of the islands of Langerhans.

Twenty-two revealed sclerosis of the islands of Langerhans.

Five revealed sclerosis with hyaline degeneration of the islands.

Ten revealed arteriosclerotic changes.

Two revealed complete necrosis of the islands of Langerhans.

One revealed amyloid changes.

Three revealed no pathologic changes in the islets.

It is of interest to note that the most consistent pathologic change in the islands of Langerhans was sclerosis; it was present in 46.2% of the necropsies studied. On the other hand all types of pathologic changes were present and in three cases the islets revealed no pathology whatsoever. Yet these three cases were fulminating cases and death was due to diabetic coma. What are we to infer from these statistics? Quite evidently this data throws little light on the direct cause of diabetes, at least not from a pathologic standpoint.

A study of the pathologic changes in the pancreas of fatal diabetics identical with that of the writer is reported by Cecil[4]. His group comprises 90 cases; 43.3% of his cases revealed sclerosis of the islands of Langerhans; 21.1% revealed hyaline degeneration. It is a striking fact that he found 12% of his cases showed no pathology of the pancreas as compared with only 7% in my series of 47 cases. It will be noted that Cecil's findings closely parallel those reported above. We can only say, however, that sclerosis is the most consistent pathologic change, and the islets may even be normal.

SYMPTOMATOLOGY

The symptomatology of diabetes is not, as a rule striking. Usually the first symptom the patient becomes aware of is polyuria. Coincident with this is excessive thirst. The patient will often remark to a friend or to the physician that he cannot get enough water to drink. Usually the onset is fairly gradual, progressively increasing in severity. The intake of enormus quantities of fluid and a large output are common early manifestations. There is more frequently an increase in appetite, yet in some cases anorexia may occur. Loss of weight is quite common, especially if the disease progresses for a period of time without treatment. Many diabetics, prior to the onset of the disease are quite obese; this fact, too, explains weight loss. Pruritis is common, pruritis vulvae in females, balanites and pruritis ani in males[4].

Weakness, diabetic neuritis, trophic skin changes, obstinate eczema, furunculosis and a variety of similar symptoms, will be elicited with a carefully taken history and should lead one to suspect diabetes, even before the urinalysis is done. Notwithstanding, a vast majority of diabetics are recognized coincidently during a life insurance or industrial examination, or in the course of an examination for some other suspected disorder. This fact only tends to strengthen the argument in favor of a careful history, and clinical examination of all patients by the physician.

The following case is typical of the usual onset of diabetes[7].

July 30th, 1931, a white female, age 14, of Caucasian descent, entered the office with the complaint of unusual thirst, the passing of large quantities of urine, weakness, loss of appetite and loss of weight. Duration of these symptoms, about twenty-one days. Four weeks prior to entry she had had an attack of influenza. There was no family history of diabetes. The history revealed that she had always been fond of candy, pastry, etc. Onset of puberty six months prior to entry. Her type was thin and rather frail. Urinalysis on a single specimen revealed four plus sugar. Blood sugar on fasting diet 296 mgs. per 100 c.c. blood. The patient was admitted to the hospital for treatment. Twenty-four hour specimen revealed 2.8% sugar. The patient was taught to test her own urine, and was released on an 1800 cal. diet. C. 60, P. 80, F. 120, with U XII insulin t. i. d. 20 minutes before meals. She responded nicely for the next ten days.

She was not seen again for a period of several days when she was admitted to the hospital in profound coma. A check-up on the history revealed the fact that the family was practicing Christian Science and that for one week the patient had discarded treatment for the administrations of a practitioner.

On this admission the urine revealed a four-plus sugar. The blood sugar revealed 520 mgs. per 100 c.c. blood. The C O combining power of blood was not determined. Usual measures of treatment were instituted. During a period of eight hours the patient received 290 units of insulin. She revived from the coma and was quite normal in twelve hours. Recovery was uneventful. She was discharged from the hospital in four days following an uneventful recovery. Recent investigation reveals the fact that she later discarded treatment for Christian Science and suc-

cumbed to a diabetic coma in a neighboring city.

DIAGNOSIS

The diagnosis of diabetes mellitus as previously pointed out depends primarily upon the finding of sugar in the urine. Glucose is not a normal constituent of the urine[4]. Its presence is indicative of an abnormal condition existing in the body. It may be benign, as observed in alimentary glycosuria, or "renal diabetes." On the other hand it may be inductive of true diabetes. Again it may be associated with non-diabetic conditions such as hyperthyroidism hyperpituitarism and other disorders, serious but nevertheless essentially non-diabetic.

A. PHYSIOLOGY OF CARBOHYDRATE METABOLISM

If one considers carefully the physiology of carbohydrate metabolism, it will enable him to better understand the value of clinical and laboratory data in making a diagnosis of diabetes. One hundred per cent of all injested starch is converted into glucose. Fifty-seven or fifty-eight per cent of protein is converted into sugar[4]. Ten per cent of fat is available glucose. The normal fasting blood sugar varies between 80-120 mgs. per 100 c.c. blood (Folin-Wu). The urine is normally sugar free.

There is two definite phases of carbohydrate metabolism; that concerned with the storage and utilization of glucose by the liver and that by the tissues. The role of the liver is vitally important. Sugar is absorbed from the intestine in the form of glucose and carried by the portal system to the liver. A portion of this is utilized directly by the liver, the process being known as glycolysis. That which is not utilized is converted into glycogen and stored. This process is known as glycogenesis. The normal liver is capable of storing 150 to 200 gms. of sugar[4]. The stored liver glycogen serves as a supply to maintain the normal blood sugar level. Glycogenolysis, or the reconversion of the liver glycogen into glycose occurs through the action of an enzyme, other than insulin, although it is probable that insulin also plays a part in this process.

The important role of insulin is believed to occur in tissue glycogenolysis, i. e., the conversion of the glucose in the blood into tissue glycogen, in which form it is capable of being utilized for the carrying on of the metabolic processes of the body. As in the liver there is stored in the tissues a glycogen reserve; this process being designated as tissue glycogenesis. It is through these two very important mechanisms that a sugar reserve is maintained and a constant available supply is present in the body.

One can readily see that any condition affecting the storage or utilization of sugar in the liver or tissues will definitely affect carbohydrate metabolism. Serious liver damage as well as insulin deficiency is then a factor. Another metabolic influence is worthy of mention, epinephrine.

Epinephrine[8] or adrenalin has a very pronounced affect on increasing the amount of sugar in the blood, either by more rapidly releasing glycogen from the liver or by inhibiting the extraction of glucose from the blood by the tissues[8]. A rise of 35 to 49 mgs. of sugar per 100 c.c. blood will occur in forty-five minutes following the administration of M. X. of 1 to 1000 adrenalin chloride[8]. This phenomena is perhaps a vital protective mechanism at times in the prevention of sudden hypoglycemia in normal individuals as well as diabetics following an over supply of insulin.

It is apparent that there may be all degrees of severity in diabetes. Physiologically the seat of trouble might be in the liver, in tissue utilization or even in the kidney. The term alimentary glycosuria has been coined to explain the presence of sugar in the urine during the process of digestion, and not at other times. There is considerable argument as to whether alimentary glycosuria is a benign condition or one which should be regarded with no little concern. Ringer[9] states that he has reached a point where he considers all his alimentary glycosurias as mild diabetics. Certainly they merit close observation over a considerable period of time. What tests should one employ to substantiate the urinary findings. Unquestionably the blood sugar is reliable. A relative increase in the blood sugar proportionate to the glycosuria is quite suggestive that diabetes exists.

B. GLUCOSE TOLERANCE TEST

The glucose tolerance test is an excellent guide. Several methods have been devised for the carrying out of this test. The injestion of 1.75 gms. of glucose per kilogram body weight will show a rise in the blood sugar of 40-50 mgs. per 100 c.c.

blood, during the first one and one-half hours following the taking of the glucose[8]. From one and one-half hours to three and one-half hours the blood sugar recedes to normal. Coincident with the blood sugar findings there may be a glycosuria. Blood and urine specimens should be taken every thirty minutes for four hours. For practical purposes this test can be modified to the injestion of 100 grams of glucose and the determinations carried out as described. This procedure is without question a valuable one in dealing with the alimentary or renal type of glycosuria, or in the border line case, in which it is doubtful as to whether the patient should be strictly dieted or allowed a moderate range of freedom.

It is my firm belief that fewer cases of diabetes would be overlooked if the physician availed himself of the glucose tolerance test. I candidly admit that I have utilized this test very little during the past four years, in dealing with a considerable number of diabetics. At present I have three cases under dietary management, who are excellent subjects for this test. I do, however, employ the blood sugar determination and the twenty-four hour urine specimen extensively. In our clinic all suspected cases of diabetes get frequent blood sugar determinations. For the past two years I have personally done all the blood sugars. We employ the original Folin-Wu method.

RENAL DIABETES

Renal glycosuria or renal diabetes is intended to designate a condition of either normal renal threshold with a hyperglycemia of the blood or a low renal threshold with a normal blood sugar[8]. In either instance glycosuria may occur. The normal renal threshold lies within the limits of 160-180 mgs. A case is recorded wherein the blood sugar rose to 350 mgs. per 100 c.c. of blood during an ether anesthetic, yet no sugar was found in the urine. There can be no doubt but what some normal individuals possess an increased permeability of the kidney to sugar. The term, renal diabetes should be used with caution. Only after repeated blood sugars, twenty-four hour specimens and tolerance tests is one justified in making a diagnosis of renal glycosuria.

The following case illustrates very well a glycosuria, probably intestinal or renal, but nevertheless meriting close observation over a long period of time[8].

A white male, age 9, always well and healthy and the son of a physician, develops a quite noticeable polyuria. Single specimen of urine following his usual meal reveals one plus sugar. Twenty-four hour specimen reveals slightly less than one-plus sugar. Single fasting specimen, negative. Blood sugar, fasting, 96 mgs. per 100 c.c. blood. No other symptomatology. Family history negative. The boy is placed on a moderately restricted diet and repeated specimens of urine are sugar free. There is also a definite improvement in the polyuria. Apparently this case is one in which there is a spilling over only at the height of digestion. In this instance correction of diet is the solution. The glucose tolerance test while indicated has not as yet been done. I am of the opinion that this patient deserves close observation throughout his life.

THE DIFFERENTIAL DIAGNOSIS

The differential diagnosis usually presents few difficulties. It is only necessary to bear in mind those conditions which give rise to glycosuria but have only in few instances a direct association with diabetes. Alimentary glycosuria and renal glycosuria have previously been discussed and need no further elaboration here. The well known discovery of Claude Bernard, that puncture of the floor of the fourth ventricle gives rise to glycosuria is of more academic interest than of clinical importance[10]. Griffin and Bowles in 1923 showed a fairly consistent association of diabetes with pernicious anemia[11]. Starling has produced potential diabetes in animals by the action of phloridzen[10]. Apparently, the drug gives rise to an increased permeability of the renal epithelium.

Generally, it may be said that glycosuria may occur in case of altered function of the island of Langerhans in the pancreas, hyperactivity of the suprarenal glands, hyperactivity of the thyroid gland, or of the posterior lobe of the hypothesis[8].

One might ask, why or in what way does hyperthyroidism affect carbohydrate metabolism? It is a well established fact that glycosuria is frequently encountered in the thyroid patient[8]. This is explained by the action of thyroxin on the liberation of glucose from the liver. It produces a hypersensitiveness on the part of this organ, thus converting glycogen into glucose more rapidly than normal. The glucose is piled up in the blood stream and spilled

over into the urine. Rises of five or ten per cent have been observed in toxic patients. It is obvious that unless an abundant supply of starch is taken by the individual suffering from thyrotoxicosis, the glycogenic store in the liver will be depleted and a subsequent hypoglycemia will occur.

The exact reverse of this condition is found to occur in hypothyroidism, myxodema or cretinism. Many of these cases carry a blood sugar as low as 60 mgs. per 100 c.c. blood[3]. In Addison's disease hypoglycemia is a most consistent finding due to the depletion of epinephrine. In hypopituitarism we usually encounter a state of hypoglycemia. This depends somewhat upon the lobe involved and whether there is an associated thyroid factor. Hyperpituitarism, involving the posterior lobe of the pituitary, i. e., acromegaly and correlated states of over activity have a tendency to produce a hyperglycemia. Cushing and his co-workers have established this important fact[3]. The action of this mechanism is such that the patient might be incorrectly diagnosed as a diabetic. The action is analagous to a true insulin deficiency in severe cases. These posterior lobe hyperactive cases are probably normally supplied with insulin but the oversecretion of the posterior lobe is antagonistic to its action thus preventing its maximum activity in tissue glycogenolysis. Such cases are then not in any sense true diabetics. Treatment of the pituitary condition whether it be one of hypo or hyper dysfunction invariably clears np the glycogenic disorder.

Of considerable interest to the surgeon is the affect of ether and chloroform anesthesia on carbohydrate metabolism. It has been demonstrated that patients under ether anesthesia have had a rise in blood sugar of seven to eight mgs. of glucose per 100 c.c. blood for every ounce of ether administered. Trumper and Cantarow[4] report cases in which the blood sugar has attained the height of 400 mgs. per 100 c.c. blood during the course of a prolonged anesthetic. It is thought that the creation of a temporary acidosis by ether, inhibits the action of insulin, thus bringing about this rise. The degree of rise in blood sugar depends upon the duration of the anesthesia, the amount administered and to a lesser extent upon the individual's reactivity to ether. In those cases followed by blood sugar determination during the course of an anesthetic the blood sugar has been found to fall to normal or to somewhat below normal within twelve to fifteen hours with no untoward effect to the patient.

Asphyxia[5], due to the production of a state of acidosis markedly inhibits the effect of insulin on tissue glycogenesis, thus creating a rapid rise in blood sugar with a subsequent glycosuria.

It is only through a thorough understanding of acidosis, that one is able to appreciate the mechanism at play in the insulin fast patient. It requires the equivalent of 60 gms. of carbohydrate to burn up the fat in the process of digestion and assimilation[1]. Therefore in the case of the diabetic where there is little if any available carbohydrate, these ketone bodies pile up in the body. There is as a result of this a calling on of the alkali reserve; acidosis or ketosis is the end result. For this reason in balancing a diet the ketogenic-anti-ketogenic ratio must be consid-ered. Insulin has an optimum affect at a p. H. 6.5. The p. H. of the blood is normally 7.32. One can readily see that in case of acidosis the maximum effect of insulin is not achieved. This fact readily explains the large doses often required for the coma patient.

The differential diagnosis of coma rarely causes difficulty. Of course hypoglycemic shock is important and is often confusing clinically. Of lesser importance are uremia, cerebral hemorrhage, diabetes insipidus, meningitis and chloral and morphine poisoning[2].

CASE REPORTS

The following case illustrates some of the confusing problems which arise in diagnosis[6].

A Jewish woman, age 60, was admitted to the medical service of the Peter Bent Brigham Hospital, Boston, 1927. She was apparently in profound coma. She was extremely ademato us. Urine revealed four plus sugar, two-plus albumen and was loaded with hyaline and granular casts. Blood sugar, slightly above 300 mgs. Blood urea nitrogen 56.0. Treatment was instituted for diabetic coma. Recovery was slow. However, she was released six days after entry definitely improved. Seven days after this admission she was re-admitted in coma, again markedly edematous and died in a few hours, in spite of all measures. Was this patient primarily diabetic or renal? Eye grounds ex-

amined at first entry revealed an extensive albuminuric retinitis. A diabetic retinitis was not recorded. Post mortem was not obtained.

A case of diabetic coma with asthenic uremia is reported by Marchbanks and Graham in November, 1932[12].

A rather confusing case was admitted to the medical service of the Kansas City General Hospital in January, 1913. A white male, age approximately 60. He was in a state of coma on entry and was placed on energetic treatment. The urine revealed four-plus sugar. Blood sugar 316 mgs. He showed a very poor response to all measures employed. This patient gave a history of known diabetes of about four years duration. He showed persistent albumin and casts in the urine with a non-protein nitrogen persistently above normal. The eye grounds were suggestive of albuminuric retinitis. He had a carbuncle on the back of his neck that showed practically no improvement after thirty days treatment. He at no time carried a normal blood sugar. The urine was seldom sugar free. Hypoglycemic reactions were frequent.

This case unquestionably was one of arteriosclerosis and chronic nephritis, as well as diabetes. As to which disease played the major role is difficult to say. The high incidence of arteriosclerosis and nephritis in the older diabetics is well known.

SUMMARY

The diagnostic criteria of diabetes are relatively simple if the physician will avail himself of them. It is probable that diabetes is not on the increase, but rather is being better diagnosed than formerly. A better conception of this disease is gained if we review the physiology of carbohydrate metabolism. The urinalysis is without question the important factor in making a diagnosis yet other facilities, namely the blood sugar, the glucose tolerance test, history and clinical examination are safeguards against the pitfalls that can easily be avoided.

Disease of the thyroid, the suprarenals, the pituitary and the pancreas, is capable of giving rise to glycosuria. The benign glycosurias are open to question as to the role they play in carbohydrate metabolism. It seems that there is little to gain from a study of the pathology of the pancreas. Further endeavor in this field may yet throw more light on the etiology. Confusing cases from point of view of diagnosis are more the exception than the rule.

CONCLUSIONS

1. Diabetes is at present diagnosed more frequently than a decade ago.

2. The urinalysis does not give a true index to defective carbohydrate metabolism.

3. The pathology of the pancreas is of minor significance in diagnosis.

4. The history, clinical examination, blood sugar and occasionally the glucose tolerance test are indispensable aids to correct diagnosis.

5. Glycosuria is almost predominantly associated with dysfunction of four glands of internal secretion, namely, the pancreas, the thyroid, the pituitary and suprarenals.

6. The diagnosis of alimentary glycosuria and renal diabetes should be made only after exhaustive study.

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DIABETES IN CHILDHOOD

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Diabetes is one of the oldest of recorded diseases. Its symptoms were described in the Ebers Papyrus about the time of Moses. It was named by Araeteus who likened it to a syphon, hence we get its Greek name, Diabaino. Paracelsus performed the first chemical experiment in that he found the evaporated urine con-

tained sugar. The ancient Hindus noted the sweet taste of the blood and urine, but through Willis it was brought to the western world. The distinction between diabetes insipidus and mellitus was called attention to by Darwin in 1778, and so on in the evolution of its present knowledge.

But the knowledge so gained has had no practical bearing on the treatment of the diabetic patient, especially the children. A child being so afflicted was doomed to an early and sure death. The younger the patient the shorter the duration of life, hence the clinical observation of cases then presented the coma picture, for the child did not live long enough to have the many complications which a child so afflicted now has. The final picture was noted in a slowly developing non-livid coma. The paucity of the cases in early literature is also a matter of wonderment, as Pavy reported eight cases under ten years in 1860, and Prout one child in seven-hundred cases, while Priscilla White reports seven-hundred fifty cases in Joslin's clinic.

So it is now more common than is generally suposed. The disease itself has risen in mortality statistics from twentieth place to tenth place at present, and then the alertness and intelligence of the physicians today may have something to do with diagnosing more cases. Many cases are overlooked because it was commonly believed to be a disease of late adult life, and rarely occurring in infancy or childhood[1]. "The number of cases of diabetes with onset in childhood parallels the number with onset in old age, i. e., there are as many children who have diabetes before they are ten, as there are men and women who have onset after they are seventy." Sixteen per cent of these juvenile cases had onset of the disease under the fourth year of age.

The ages of greatest incidence are the same as those of greatest growths, i. e., six to eight, and twelve to fourteen years. This may give some reason to think that the endocrines may be a causative factor, especially those having to do with growth and maturity. The sexes seem to be about equally afflicted in childhood, yet in adults, women have been found to be affected more than men. The diabetic girl matures later than her non-diabetic sister, and if not properly managed after the catamenia is established, she will quickly notice an absence of her regularity. The male diabetic often loses his libido[2]. "Diabetes in infants one year of age is rare, and even with the addition of their own cases, Joslin and White collected but thirty-one cases." Congenital diabetes is practically unknown.

Why do you stress the case of the diabetic child? The reason is very obvious that because with the discovery and practicability of insulin by Banting in 1922, it has given a method of handling these patients which has been the outstanding contribution to medicine that has occurred in my lifetime. In eleven years it has snatched these children from a certain death in from six months to two years, to strong vigorous youngsters, normal in weight and mentality. The well treated children grow as fast and mature as normally as their siblings, collaterals, friends or relatives who are not so handicapped, and to watch the growth and well being of these youngsters as compared to the utter hopelessness of the former management, makes me exult in the fact that I am a physician and see the effects of this magic touch to one so afflicted.

Now diabetes in childhood has a different underlying pathology of the pancreas than in the older diabetic, and the pathological changes in the islands of Langerhans are very few. Warren[3] claims the lymphocytic infiltration is the most characteristic lesion. He claims in most cases the islands are normal from a qualitative standpoint as well as normal in numbers and histology, and most important of all, the children should have advantage of the regenerative powers and stage a comeback more than the adult. Boyd also stresses this fact. The pancreas has regenerative power not unlike that of the liver, the histological findings of normal B iletin cells show certain granules when normal, and disappearance of those granules when the gland is fatigued. Warren says, "perhaps the simplest physiological analogy of the pancreas in diabetes would be the thyroid in myxodema, yet in the thyroid cells no such anatomical change develops." In the adult or senile diabetic the pathological changes may be so great that it causes the pathologist to wonder why the gland functions at all. The iletin glands of the pancreas begin to function about the fourth month of the embryo, long before the acinous glands begin to take up their activity. This has given current to a theory that the pancreas of a foetus of a pregnant diabetic

assists in furnishing the insulin to a mother during the latter stages of her gestation, and increases her ability to metabolize carbohydrates, a theory which has not held good in our clinical experience. It is a fact however, that the new born most always dies soon after delivery in a state of hypoglycemia because the excessive load is taken off the hypertrophied pancreas causing a profound hypoglycemia unless carbohydrates are generously given it at first. (Hyperinsulization).

Now let us see why one child develops diabetes and another does not, or why the potential diabetic? The basis on which inheritance rests is bound up in the chromosomes, which are bearers of hereditary factors or genes. Dominant factors may be brought out in mathematical accuracy, while recessive characters on the other hand, may be potentially present in a given stock for generations, and never be manifest. Doctors Joslin and White[4], in order to answer this question of eugenics have made this dogmatic statement, "if a diabetic marries a non-diabetic with a diabetic heredity, half of the children should develop it; if two non-diabetics, but each hereditarily predisposed to diabetes marry, one-fourth of the children should get it; but if a diabetic marries a non-diabetic who is without a diabetic heredity, no diabetes should appear in the offspring." In other words diabetes is recessive rather than dominant. This observation has become manifest since the use of insulin because it lengthens the lives and makes them more normal. This prolonging life has also allowed the more searching family history, so that Joslin has found the element of heredity in fifty per cent of his children. Oliver Wendell Holmes says that we are all omnibuses in which all of our ancestors ride.

When one considers the noninfectious nature of diabetes, the occurrence of the disease in twins, identical and fraternal, it points to a hereditary background or to some congenital liability to functional failure in the islands of Langerhans, then it strengthens our point. Curtis[5] claims that in the recessive type it occurs more commonly before the age of forty, is generally grave at the onset, and tends to be self-exterminative. The dominant type is more frequently encountered in elderly people, and almost invariably is mild and does not materially affect the general health.

Curtis in the study of thirteen sets of identical twins found that quite a few developed diabetes, almost simultaneously, even though reared apart and under different conditions, and adds that when one develops diabetes, the other should be carefully watched so as to not strain his carbohydrate metabolism, in other words, he is a potential diabetic.

"Superiority is more characteristic of the potential diabetic child than is defectiveness[6]." At the onset of the disease the diabetic child is precocious, it takes great interest in its trouble, and glories in the fact that it can compute its diet, and want to help others in arranging their diet. "His physiological and roentgenological ages are more than twelve months in advance of his chronological age, and his mental development is above the average by six months." He is taller of stature than his siblings or collaterals, he matures and develops in a normal manner, holds his own in classroom and sports when properly treated with adequate nourishment, and sufficient and well spaced insulin. What height is to children, obesity is to adults as potential factors in diabetes.

The baby born to a diabetic mother is nearly always a large baby, weighing over eight pounds, and it is found that cesarean section is the method of delivery in primipara being attended with less hazard to the baby and with less shock and danger to the mother. This has been our experience, and all recent literature, both of obstetricians and internists, bears out this fact.

As to racial predisposition, Priesal and Wagner found that twenty-seven per cent of their cases in one-hundred eight diabetics, with onset in childhood had hereditary tendencies, and that this heredity was forty-three per cent in Jewish children as compared with twenty-one per cent in Gentiles.

"It seems to be well accepted that diabetes even in a fulminating form can follow infectious diseases, such as typhoid fever, scarlet fever, influenza, etc. This has recently been emphasized by Williams and Dick[7] in the McCormick Institute for Infectious Diseases in Chicago, claiming that in infectious and contagious diseases half the patients will develop a glycosuria if given enough glucose. Likewise in experimental infections in animals, a similar transient glycosuria is produced. This is

accompanied by an increase in the blood sugar concentration while fasting, and by alterations in blood sugar curves as shown by dextrose tolerance tests in animals." Geyelin[8] reports eight cases of five weeks duration following infection. White[9] in a study of one hundred diabetic children, found only two who did not have a history of an acute infection preceding the onset of diabetes. True it is that potential diabetic or low grade diabetic will be made much worse during an acute infective disease, even so much as a common cold, and insulin will have to be increased during that period. But when once diabetes is established then infections have a profound effect on the lowering of sugar tolerance, production of ketones and possibly coma, and the likelihood of running a riotous course, so these unfortunates should be carefully handled during the common colds, sore throats and boils, and insulin dosage increased, but let the infection be removed by the crisis of a pneumonia, the removal of a bad appendix, or amputation of an infected gangrenous arm or leg, and the dosage of insulin needed will fall dramatically and return eventually to a preinfective stage. There is no such thing as insulin fast cases, i. e., where insulin in proper dosage is not effective, but in the presence of pus or bacterial infections the dosage may have to be increased to where you shudder at the amount. Let me remark here that the shortage of glycogen in liver and muscles will be depleted so that hyper and hypoglycemia may be present in very short space, necessitating a much closer scrutiny of your patient during those trying times.

Bowman[10] in his article deduces: 1. Heredity plays little part in etiology; 2. That the appearance of the diabetes usually follows an acute illness such as scarlet fever, measles, mumps, or cystitis.

The development of diabetes in children is associated with a more definite time than in adults, often it comes on with sudden onset without any premonitory symptoms, but, as it were, springs full blown into existence, again it may come on more insidiously and the child is noticed to be losing flesh, is less active at play, urinates more frequently and copiously. There may be an associated increase of appetite and desire for water. This early observation may be due to the careful supervision of a mother or nurse. The child is taken to its physician, who examines its urine and finds sugar or possibly acetones and diacetic acid. A further study of the blood sugar while on its accustomed diet, shows an increased amount of glucose above 120 mgm. per 100 c.c. of blood. A further study may show that the sugar curve after a meal of glucose, rises to a higher point and does not come back to its level of the fasting stomach in three hours when measured at hourly intervals. This is a questionable procedure however. The child is observed while put on a maintenance diet of possibly two or three grams of proteids, per kilo of body weight with the carbohydrate ratio of 1.5 gms. per kilo and fat to make up forty calories per kilo. Should the child continue to spill sugar on this diet, we find the amount of glucose in the two twelve hour specimens of urine, and start with insulin computing one unit of insulin will care for two grams of glucose. Or by the method of trial and error we use 5-15 units of insulin before meals, gaging the amounts on the size of the patient and on the reduction of the Benedict's solution, whether red, yellow or green. We find the larger dose of insulin needed before breakfast, as the previous day's insulin has been destroyed, during the night. In severe cases or in those who have depleted their glycogen reserve, it is even necessary to give a dose at night, with the food carefully weighed and the insulin properly spaced, and dosaged, and the patient is soon rendered sugar and ketone free.

Exercise should be as carefully dosaged as diet, as exercise causes insulin to be more effective than at bedrest. On rainy days with quietude in the house, the patients need more insulin than on bright days when they can romp and play. This is a point that cannot be too much emphasized. A run around the yard before breakfast and the engaging in moderate exercise will assist immeasurably in the efficiency of insulin. In fact I have seen many insulin reactions occur from too much muscular activity on the same diet, and insulin, that had formerly been safely tolerated.

Emotional disturbances, next to exercise have to be computed as a disturbing factor in sugar metabolism, for it lessens the ability to store glycose, possibly due to the effect of the suprarenal secretions on the pancreas. The proper care and guidance of an intelligent mother or nurse with suitable nervous hygiene bespeaks cooper-

ation of the child and presages a happy outlook, especially up to the school age, and even then the early training has much to do with the adherence to a proper regime when away from her. The fool always dies young. These patients should be encouraged to live a normal existence and not be obscessed with the idea that they are different from other children, and bound by limitations of physical or mental exertions. One of my boys is a star player on a basket ball team and there is nothing effeminate about him.

Now with the diagnosis of diabetes established it behooves them to stick with the treatment assiduously one day after another, because a well treated case in the youth generally increases tolerance from week to week and the possibility, nay even the probability, of his getting to the point of going without insulin and even eating more liberally is to he held as an incentive. However, going on sugar sprees or leaving off insulin even for a short while will most likely lower his sugar tolerance, possibly precipitate coma, and make him worse for quite a while, laying him open to many complications which are in store for an inadequately treated case.

The first complication which we shall narrate is the most common and most dramatic, and that is coma. Uncomplicated coma is preventable and curable in the juvenile, but it increases in gravity and lessens in incidence in adolescence until it attains a fifty per cent mortality in the quinquenium". Of the total number of comas treated in the hospital, children represented 89, and of these there was but one death. What a contrast to the period prior to the discovery of insulin, when all diabetic children died of coma. Forty-four (⅔) of Joslin's cases who have died outside his hospital have died of coma. There are more deaths under ten years of age, while in the second decade the mortality was only three per cent in comparison with 13.1 per cent in the whole series. Ten to twenty per cent of diabetic children have coma at one time or another, so one must be continually on the watch for it. Complicated coma such as is brought on by acute infectious diseases, boils, colds and pus infections, is more serious and demands more constant attention with some one person to direct and guide the management, not unmindful of his responsibilities, nor leaving his patient until the coma has been relieved. Joslin's trained crew for the care of this complication is ideal, but I have even seen a lone physician in the country stay with his patient until he was out of danger.

Gottschalk of Stettin demonstrates that hospital admissions for diabetic coma can be reduced to zero in a city of 270,000. This took educated and trained families as well as physicians. In managing a case of coma, conserve body heat by blankets, dehydration is then to combat, normal salt solution either intravenously or by hypodermaclysis or intraperiotoneally and repeated until possibly 6000 c.c. in twenty-four hours are given, lavage of stomach because of the dilatation and vomiting, enema to cleanse the rectum, insulin given according to reaction of urine to Benedict's solution at one-half to two hour intervals, five to forty units after hourly tests, or to blood sugar estimation, sugar to be given if urine scant or as a buffer for insulin. No alkalies. Cardiac stimulants are rarely needed in children, but adrenalin or ephedrine if there is circulatory failure. Intravenous fluid given slowly and freshly triple distilled water in clean apparatus with glucose out of freshly opened ampoules.

Coma must be contrasted with insulin shock and surgical abdomen, most likely appendicitis.

Coma has a slow onset. Insulin shock rapid. Coma due to dietetic error, infection or lack of insulin. Insulin shock due to too much insulin, too little diet, or too much exercise.

Coma, skin dry and florid, insulin shock, moist and pale.

Coma, Kussmal's breathing, i. e., hyperpnaeic, non livid air hunger. Shock breathing, normal or shallow.

Coma, pulse weak and rapid. Shock, is full and bounding in spite of collapse.

Coma, vomiting preceding unconsciousness, abdominal rigidity and distension. Shock, vomiting rare.

Coma, convulsions rare. Shock, convulsions and bizarre nervous phenomena the rule. Sehestedt" states that in insulin shock, particularly in actual unconsciousness, the Babinski signs are frequently positive on one or both sides. He has never found a positive in diabetic coma, nor seen a statement to that effect. Joslin claims to have not found it in either condition.

Surgical abdomen as appendicitis may

precipitate and complicate coma, both have leucocytosis and polynucleosis, both vomit, both have tender rigid abdomen, and your diagnostic acumen will be taxed to its limit.

The indirect complications of diabetes as arteriosclerosis, cataract, etc., are not present in adequately treated cases, but come on in children when they are not carefully handled and do not cooperate with the management. Hence age does not negate these indirect complications.

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DIABETES MELLITUS AND ITS RELATION TO VASCULAR DISEASE

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The relation of diabetes to vascular disease, or if you prefer, diabetes complicated by vascular disease, is perhaps the most important problem confronting the diabetic. Today the greatest single cause of death in diabetes is arteriosclerosis in all of its ramifications. The diabetic population has a higher percentage of arteriosclerotic complications and develops them at an earlier age than do non-diabetic individuals. Since the advent of insulin and the knowledge pertaining to its use, death from coma in the absence of an infection can be almost entirely prevented by the intelligent co-operation of patient and physician. As result these people live longer, thus allowing them time for the development of vascular disease and projecting more of them into the age period when vascular disease is prone to develop.

ETIOLOGY

Obviously a final answer to the question of etiology will depend upon the solution of the ultimate cause of uncomplicated arteriosclerosis. Why it occurs so much more commonly in diabetes is still unknown. There have been attempts to correlate it with obesity, hyperglycemia, and acidosis. No adequate proof has been adduced to support any of these contentions. Perhaps the most promising field of investigation at the present time is into the disturbed fat metabolism with its elevated blood fat and blood cholesterol. We know that arteriosclerosis can be produced experimentally by feeding a diet high in cholesterin and we know that diabetics carry a hypercholesterolemia and the possibility of a causal relationship must be considered.

INCIDENCE

We know that arteriosclerosis occurs more commonly in men than in women. Although this is also true in the diabetic the difference is a comparatively small one. Particularly is this equalizing influence of diabetes seen in the case of coronary sclerosis. The age incidence of arteriosclerosis is also an interesting study. In the general population the incidence of arteriosclerosis increases with age. In the diabetic, not only is this true, but arteriosclerosis also increases with the duration of the diabetes. As a general rule, regardless of the youth of the patient, a diabetes of five years or more duration will produce arteriosclerosis. The longer the duration of the diabetes the more extensive will be the arteriosclerosis and the higher will be the percentage of deaths due to some form of arteriosclerosis.

CORONARY DISEASE

Of all the arteriosclerotic deaths in diabetes heart disease accounts for the greatest number. There are two main types: (a) coronary sclerosis with or without infarction, and (b) hypertensive heart disease. Of these coronary disease is by far the more important. Coronary disease has a much higher incidence in the diabetic than in the non-diabetic. Nathanson[1] in an analysis of one hundred autopsies on diabetics found severe coronary disease in 41% of diabetics of all ages, and in 52% of diabetics over the age of fifty, as compared with 8% in his control series. These figures include coronary thrombosis, coronary sclerosis with myocardial insufficiency, and asymptomatic cases. Warren[2] found that 12% of his series of autopsied diabetics died of coronary thrombosis. Root[3] in a pathological study

based on 80 autopsied diabetics found coronary sclerosis present in some degree in every individual who had had his diabetes five years or longer. Coronary thrombosis, including old and recent infarction was found in 25%. Blotner[2] has a similar series of 77 fatal cases of diabetes. He found that 35 showed well marked disease of the coronary arteries, eight died of cardiac infarction, and 33 died of some type of arteriosclerotic heart. In the 35 cases of coronary sclerosis demonstrated pathologically a definite hypertension was present in 15. Expressed in a different way, Nathanson found that of 24 autopsied cases of coronary thrombosis six were diabetics. In Levine's[3] series of 145 cases of coronary thrombosis, diabetes contributed 34 or approximately 25%. In the general population the incidence of coronary disease in males and females follows approximately a 4:1 ratio. In diabetes there is a marked levelling influence so that the ratio is approximately 3:2. A rather interesting point brought out by Nathanson[1] is that diabetics with gangrene have a higher incidence of coronary disease than other groups. There appear to be two precipitating causes of coronary thrombosis peculiar to diabetes — namely acidosis and insulin hypoglycemia.

The signs and symptoms of coronary disease are no different in diabetics than in others and it is beyond the scope of this paper to discuss these points. However, I believe that some discussion of treatment is worthwhile. Ordinarily in treating a diabetic we desire to keep him sugar free and to keep the blood sugar in the normal or near normal range. However, in treating arteriosclerotic diebetics and especially is this exemplified by the patient with coronary disease, this theoretical ideal must be radically altered. These patients have become accustomed to and require a blood sugar at a higher level in order to function properly. If we subject the heart with diseased coronary arteries to an artificial lowering of the blood sugar it may respond with angina or signs of myocardial weakness. Similarly the arteriosclerotic brain will respond with a wide variety of mental symptoms. Coronary thrombosis has been reported in a number of instances immediately following an insulin injection suggesting an etiological relationship. Anginal attacks would seem to be easily explained; actual thrombosis would be more difficult to explain. Treatment of the arteriosclerotic diabetic must be highly individualized. Many do satisfactorily untreated because of the mildness of the diabetes. The majority can be handled with diet alone. When insulin is necessary it should be used with great care, the patient should be kept under close observation, and the urine and blood closely followed. Insulin therapy should be started with small doses gradually increasing if necessary. For each one of these patients the blood sugar level should be found which will allow that patient to function at his best. This level will usually be in the neighborhood of 160-180. Almost all of these patients have a high renal threshold and consequently can be kept sugar free even with a high blood sugar. The following table summarizes the pathological findings regarding coronary disease in the diabetic.

	Autopsies	Per Cent of Severe Coronary Disease	Per Cent of Coronary Thrombosis	Deaths From Heart Disease
Blotner	77	45	10	43
Nathanson[1]	100	41		
Root	80		25	
Warren	282		12	
Total	539	43	14	43

GANGRENE

Gangrene is another important manifestation of vascular disease in the diabetic. The causes of the vascular disease are the same in the diabetic as in the nondiabetic, but their relative frequency will differ. The great majority of cases of diabetic gangrene are secondary to arteriosclerosis. Some authors attempt to differentiate diabetic from arteriosclerotic gangrene but I believe that evidence for such a difference is poorly founded and that the consensus of opinion is against such a distinction. However, it is true that diabetics nearly always have the wet type of gangrene while the purely arteriosclerotic individual is more likely to have the dry type. This may be due to the high sugar content of the diabetic tissues favoring infection rather than a difference in the vascular disease itself Evidence of disturbance of the circulation may be found in 15-20% of diabetics. Such patients are in imminent danger and careful prophylactic measures should be instituted. Approximately 6% of diabetics have gangrene, and it is a contributory cause in 20-25% of all diabetic deaths. Embolic gangrene and Buerger's disease also occur in the diabetic but are relatively unimportant. The treatment of gangrene is surgical amputation in nearly

100% of cases. The operative mortality is still high especially when the process is spreading. The life expectancy of the diabetic even after recovery from an amputation is only 2-3 years so that at best the prognosis is poor[1]. On the other hand a great deal can be accomplished in a prophylactic way with an intelligent patient. I believe that every diabetic should be instructed in the care of the feet. The starting point in most cases of gangrene is an apparently insignificant injury or infection. Ill fitting shoes may cause blisters and must not be tolerated. Corns should never be cut by the patient. Ingrown toenails should be treated by the physician. Cases of epidermophytosis should receive careful attention despite their seeming harmlessness. And perhaps most important of all, the feet should be kept scrupulously clean and shoes as well as stockings should be frequently changed. With such simple measures as these great inroads could be made into the present morbidity and mortality from diabetic gangrene.

HYPERTENSION

In using the term hypertension I am referring to so-called primary or idiopathic hypertension secondary to or at least coexistent with arteriolar disease and excluding hypertension secondary to known causes such as true nephritis, aortic insufficiency, hyperthyroidism, etc. Used in this sense we may regard hypertension as a compensatory building up of hydrostatic pressure to force blood through diffusely narrowed arterioles. There are three different, although frequently associated manifestations of the hypertension state—(a) congestive heart failure, (b) cerebral hemorrhage and, (c) arteriolar nephrosclerosis. Hypertension occurs more frequent in the diabetic. John[10] states that up to the age of 50 hypertension is no more frequent in the diabetic but that above 50 its incidence becomes progressively greater. Adams[9] in an interesting clinical study compared 1001 diabetics at the Mayo Clinic with a series of controls consisting of both accepted and rejected life insurance applications. He found that the diabetic was six times as likely to have a diastolic blood pressure above 100. On the other hand he was about 6-7 times as likely to have a low blood pressure. Using averages he found no significant difference between the two groups illustrating the fallacy into which averages can lead. Bell and Clawson[11] in a clinical-pathological study found that in a group of 70 diabetics of all ages who came to autopsy 19 had either clinical or pathological evidence, or both, of hypertension; and in the same group, of the 40 who died over the age of 50, 17 had had hypertension. They also collected from the literature the records of 2486 diabetics of all age groups and found that 22.3% of this group had hypertension (systolic pressure over 150). Of this same group, 583 diabetics over the age of 50 showed an incidence of hypertension of 41.4%. Control series obtained from the general population show an incidence of 7-15%. Accepting the higher figure for the control group it means that hypertension is 2.7 times as common in the diabetic over 50 as in the non-diabetic. The diabetic with hypertension also has sclerosis of his coronaries to a greater or lesser degree and this is a formidable combination. In a case seen for the first time controlling the diabetes will usually cause a definite and often a marked fall in blood pressure. Both the hypertensive and the diabetic individual are apt to be overweight and weight reduction will prove a marked benefit to both conditions. Again the treatment must be adapted to the individual. There are few situations in medicine where it is more important to remember that the patient and not the disease must be treated.

Cerebral arteriosclerosis is comparatively common in conjunction with generalized arteriosclerosis. About 5% of diabetic deaths are due to hemiplegia, i. e., thrombosis or hemorrhage. Although few diabetics become definitely insane, in many of the elderly diabetics minor mental symptoms such as forgetfulness, carelessness, dulling of the intellect, nervousness, eccentricities, etc., are quite common. Here, as in coronary sclerosis, the symptoms may be aggravated by lowering the blood sugar, through the overzealous use of insulin.

SUMMARY

1. Arteriosclerosis is considerably more common in diabetics than in the general population.

2. The incidence of arteriosclerosis in the diabetic increases with age and also with the duration of the diabetes.

3. Approximately one-half of all diabetics die of some form of arteriosclerosis.

4. Coronary disease, including sclerosis and thrombosis, is the most important

cause of death from heart disease in the diabetic.

5. Gangrene on an arteriosclerotic basis is an important cause of death in the diabetic. Prevention rather than cure is the avenue of therapeutic approach.

6. Hypertension, including the milder grades, occurs about 2.7 times as commonly in the diabetic.

7. Cerebral arteriosclerosis in its milder forms appears to be quite common. However hemiplegia and arteriosclerotic insanity occurs no more frequently than in the population as a whole.

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CHRONIC INTERMITTENT DUODENAL OBSTRUCTION

According to Howard F. Shattuck and Harry M. Imboden, New York, (Journal A. M. A., March 19, 1932), chronic intermittent duodenal obstruction or ileus is generally accepted as a clinical entity. The two most frequent and important causes of the condition are, first, peritoneal adhesions or bands fixing the first and second parts of the duodenum, and, second, pressure of the mesenteric pedicle, or a sharp occlusive angle at the duodenojejunal flexure causing obstruction of the third part of the duodenum. The authors' study includes forty-six cases of this condition, eight of which came to operation. The symptoms are extremely variable and noncharacteristic but should suggest the possible presence of the real trouble in many instances and lead to careful roentgen study as a consequence. Accurate diagnosis depends largely on a careful and skilful roentgen study. The characteristic roentgen observations found in the various types are described. The great majority of these cases repond satisfactorily to medical treatment, but there is a small group with pronounced symptoms and marked roentgen observations which does best with conservative surgical measures.

DIETING THE MEDICAL AND SURGICAL ULCER PATIENT*

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In no field of medicine has there been greater advance in therapy in the last three decades than in the treatment of gastric and duodenal ulcers. Both gastroenterologists and surgeons have participated in the improved methods of treatment that have resulted in the brillant results that now may be expected to follow in the 80 to 90 per cent of ulcer patients who may be cured by dietary and medical management. In the remaining 10 to 20 per cent surgery should be curative except in a few of the neglected cases, and in a small proportion of the acute complications like perforations of the stomach or duodenum. Credit should be given also to nutritionists, most of whom are not physicians, who in their laboratory researches have unlocked, and placed at the disposal of the medical profession, secrets of metabolism and nutrition that perhaps represent the greatest achievement of the twentieth century for the good of all mankind.

The ulcer patient, whether medical or surgical, should have the benefit of what McCollum calls our "newer knowledge of nutrition[1]." His diet should be adjusted to his particular needs, not only as related to the proper amounts and proportions of proteins, carbohydrates, fats and minerals; its vitamin content should also be balanced. In the medical management of ulcers of the stomach and duodenum a relatively high fat diet seems best to meet the indications for the treatment of the local pathological conditions; while food rich in vitamins will build up the general state of nutrition so that the ulcer will heal and the patient become more resistant to infections of the gastro-intestinal tract[2].

It manifestly is not advisable to give the plethoric ulcer patient weighing 200 pounds the same diet as the undernourished 100 pound individual who happens to have an ulcer. The nutritional needs of the patient who has had a recent hemorrhage differ from those of the person who voluntarily leaves his work for ulcer treatment. The adolescent ulcer patient needs a relatively higher protein diet than the

*Read by invitation before the Oklahoma State Medical Association, Oklahoma City, May 16, 1933.

adult after fifty, and in many other respects the personal equation cannot be overlooked in dieting ulcer patients.

These considerations seem to be lost sight of by most physicians in dieting their ulcer patients. There are almost as many diets employed in treating ulcers of the stomach and duodenum as there are clinicians specializing in gastro-intestinal diseases. Most of the diets which are used as a routine by the medical profession generally in treating ulcer patients, are modifications of the Lenhartz[3] diet, though they are given the names of various men who have popularized them. It seems to be the usual thing to try to make a diet fit all ulcer patients, instead of preparing daily menus to suit the nutritional needs of each individual case. In other words, it appears to be the rule that when a physician makes a diagnosis of ulcer, he gives his patient some one of the diets as published in text-books without considering the patient's age, weight and general condition, or even the variable pathological states of the ulcer-bearing stomach or duodenum.

Faulty Routine Diets. In giving these routine diets no thought seems to be given as to whether the patient is receiving an excess, or a deficiency, of protein, carbohydrates and fats; and but little regard is given to the vitamin content of food. These routine diets that are given indiscriminately to ulcer patients are not always harmful immediately, though some of them for the first week contain a dangerously high ratio of fats to carbohydrates. Most of them, after the second week, contain an excess of proteins and carbohydrates and a deficiency of vitamins B and C; hence untoward remote results may occur from their prolonged use. The least criticism that can be made of the practice of routine dieting in ulcer is that it is unscientific. It is almost as important that the dieting in gastric and duodenal ulcers should be arranged to meet each individual patient's need as it is in preparing the daily menus for the diabetic.

It must be admitted, however, that many ulcer patients will get well clinically on any one of the ulcer diets provided he rests in bed for three or four weeks. It also is unquestionably true that many ulcers heal spontaneously without any effort at dieting, or at least heal to the extent that there are remissions of symptoms for weeks and months at a time. Nevertheless, a larger percentage of permanent cures of ulcers result when scientific methods are instituted for preparing daily menus suited to the nutritional needs of each individual patient.

Small Frequent Feedings For First Week. Partial physiological rest to the stomach and complete physical rest for the patient are important in the first two or three weeks in the treatment of ulcer. Lenhartz advised half-ounce feedings of an egg and milk mixture every hour from 7:00 A. M., to 7:00 P. M., increasing the amount by half ounce each day until, in six days, hourly three-ounce feedings are given. We think it better to consider the patient's habitual hours for meals and the feedings should be begun and ended about the time for his morning and evening meals provided his hours for eating do not need regulating. Eight o'clock being the average we usually begin the feedings at that hour and end them at seven in the evening. If the patient's vocation requires him to begin work early, the first feeding is at 6:00 or 7:00 A. M., and the last ten hours later.

Some of the modifications of the Lenhartz diet, notably that of Sippy[4], begin with three ounces of food every hour. This would seem to overload the stomach, which accounts for the need of using the tube for aspirating the stomach contents during the night. Beginning with half-ounce hourly feedings for ten hours and increasing the quantity by half ounce each day, with a maximum of three ounces in six days, it is rarely necessary to employ the stomach tube at night, as is the practice in the Sippy treatment.

The Use of Fats to Protect Ulcer. Lenhartz gave a high protein diet to "bind" the acid secreted by the stomach on the theory that it prevented the irritating effect of the gastric hyperacidity upon the ulcer. Rehfuss[5] has demonstrated that proteins do not bind the free hydrochloric acid in the stomach; but, on the contrary, his highest acid readings were after feeding proteins. It therefore would seem that Lenhartz's high protein diet in ulcer was based upon a mistaken conception of gastric function. Rehfuss' observations also seem to prove that the time-honored custom of prescribing meats, eggs and other food of high protein content, in gastric hyperacidity, should be abandoned.

In my original modification of the Lenhartz diet, first employed in 1906, Con-

heim's[6] theory of a deficiency of gastric mucus in ulcer was considered; and cream was added to the Lenhartz formula with the idea of keeping the ulcer coated with milk fat, thus protecting it from the irritating effects of the excess of hydrochloric acid usually present in the stomach contents of the ulcer patient. Conheim gave olive oil to ulcer patients, claiming that it promoted healing of the ulcer by protecting the exposed nerve terminals in the open ulcer. It seemed to me that milk fat would have the same effect.

Some patients objected to the raw egg mixture with the milk and cream mixture in our modification of the Lenhartz diet; and for the past few years we have been omitting the eggs. We find that the higher fat diet relieves the pain better than when the first few days' feedings contained eggs, and that the patient makes a more rapid clinical recovery. In 1906 we knew nothing about vitamins but it is known now that milk fat is rich in vitamin A, while there are no vitamins in olive oil. The addition of the cream to the milk, therefore, not only adds to the palatibility of the food formula, but is soothing to the ulcer and increases the general nutrition of the patient.

The Low Carbohydrate Diet in Ulcer. Deeks[7], in 1904, expressed the opinion that a diet excessive in sugars and starches is the most important etiological factor in ulcer, hyperchlorhydria and other gastric disorders. He, therefore, gave his ulcer patients a low carbohydrate diet and reported excellent results. Whether or not Deeks is right in his theory, all authorities on nutrition agree that the average American diet contains a great excess of carbohydrates; and since most patients develop ulcer on a diet rich in bread, sweets, rice and potatoes, it would seem advisable to change their eating habits. We, therefore, have followed the suggestion of Deeks in using a low carbohydrate diet in treating ulcer; and believe that we have been getting better results since adopting that method.

In giving a low carbohydrate, relatively high fat diet in ulcer it should be remembered that "fats burn in the fire of the carbohydrates;" and the ketogenic-anti-ketogenic ratio of Woodyatt, i. e., 1 of carbohydrate to 1.5 of fats (in grams) should be considered in dieting any individual. A higher proportion of fats may result in a mild acidosis, particularly if there is impairment of the islet function of the pancreas, which, on account of the anatomical relations of the pancreas to the stomach and duodenum, probably is not infrequent in ulcers of the stomach and duodenum. The proportion of one-third carbohydrates to two-thirds fats by calories as used by us is not nearly so high a fat diet as the Marsh Newberger diet in diabetes. When translated into grams the ketogenic-anti-ketogenic ratio in our diets averages at least 1.5 of carbohydrates to 1 of fat. The Sippy diet for the first week, i. e., one and one-half ounces of cream and one and one-half ounces of milk every hour for thirteen feedings a day, would give 199 grams (1791 calories) fat, 24 grams (64 calories) carbohydrate, and 17.5 grams (70 calories) protein, a ketogenic-anti-ketogenic ratio of about 1 to 4. This is entirely too much fat to give in one day; too high a ratio of fat for even a normal person and a dangerously high fat diet for an individual with a damaged pancreas. We have had two cases of ulcer of the stomach in which the urine showed a high degree of acetone and diacetic acid, when the patients were consuming about one-fourth the amount of fat as in the first few days of the Sippy diet. It is probable that a high degree of acidosis would have followed in these patients had they received the routine Sippy diet for the first week.

Proteins. It would seem rational in dieting ulcer patients to give no more protein than is needed for daily nutrition. Chittenden's experiments seem to show that the normal adult needs only about one-tenth his total amount of food, expressed in calories, in proteins. Others, particularly the Germans, think a higher proportion is necessary, some ranging as high as one-seventh. It is generally agreed, however, among nutrition experts, that the average well-nourished person needs no more than 75 grams (300 calories) of protein a day. In estimating our diets for ulcer patients we give the average person over fifty years of age, 60 grams protein daily, while young vigorous adults are given 75 to 80 grams. While ulcer is rare in adolescence it does occur under twenty and, if so, 100 or more grams of protein may be given a day. After hemorrhage, or if the patient is anaemic, it is advisable to give 100 or even 120 grams of protein a day. The Lenhartz ulcer diet containing 120 grams of protein a day was originally intended for

use after hemorrhage and the results were so good that it was used by him and others routinely for all ulcer patients. In dieting the ulcer patient after a hemorrhage, we use our modification of the Lenhartz diet adjusted to meet the caloric needs of the individual under treatment.

Diet for the First Two Weeks. We begin the diet of ulcer patients by giving on the first day, one-half ounce (15 c.c.) of milk (two-thirds) and cream (one-third) mixture, and increase one-half ounce each day until in six days three ounces are given every hour from 8:00 A. M., to 7:00 P. M. The proportion of two parts of milk to one of cream is maintained in all the feedings for the first two weeks. One-half ounce of strained orange juice is given at 9:00 P. M., increasing the amount by one-half ounce each day until, in six days, three ounces are given.

From the seventh to tenth day the diet is still under the basal requirement. The three-ounce feedings are given every two hours instead of at hourly intervals, with the addition of four tablespoonsful of strained oatmeal, or one-half shredded wheat biscuit, or a thin slice whole wheat bread dry toast for breakfast and supper and one ounce scraped beef and one thin slice of whole wheat bread dry toast for dinner.

From the eleventh to the fourteenth day, the diet is increased by giving a soft cooked egg for breakfast and supper and increasing the scraped beef to two ounces or substituting minced breast of chicken for dinner.

Basal Diet During Third Week. Beginning the third week and so long as the patient is in bed, he is given his basal diet, unless he is underweight, then the amount of milk and cream is increased; or if he is overweight the cream is reduced so that his own fat will be burned. If he is anaemic the proteins are increased.

The basal diet in calories is calculated by multiplying the body weight in kilograms by 25, the number of calories needed daily per kilogram of body-weight while the patient is at rest in bed. Thus the ulcer patient weighing 154 pounds (70 kilograms) would require 1750 calories a day. Of this, 60 grams (240 calories) should be of protein and the remainder (1510) should be divided into one-third (504 calories) carbohydrate and two-thirds (1006 calories) of fats; or translated into grams, 128 grams of carbohydrate and 111.6 of fats.

If an increase in carbohydrates is desired it is easily made by adding to the basal diet enough milk sugar or bread to make up the required number of grams. If the fat requirement is higher or lower than the basal diet the cream or butter is increased or reduced; and if there is anemia, enough scraped beef and cheese are added to meet the estimated amount of protein for the patient's daily needs.

Optimal Diet After Third Week. Beginning when the ulcer patient has completed his three weeks' rest in bed his optimal diet should be given, i. e., the amount needed to maintain his strength and his normal weight while attending to his daily duties. The amount needed depends upon the amount of physical exertion performed in the day. It is estimated that the average business or professional man, or the housewife, requires about 35 calories per kilogram of body-weight. Thus the optimal diet for the adult of average height and weight, 154 pounds (70 kilograms) would be 70 times 35—2450 calories. Of this, 10 per cent., 245 calories (61 grams), should be proteins and of the remaining 2205 calories, one-third, 735 calories (183.5 grams), is carbohydrate and two-thirds, 1470 calories (163.3 grams), in fats.

The laborer employed in hard work will require about 50 calories per kilogram per day. Thus the brick-layer, or the farmer, or the golfer who plays eighteen holes a day will need about 3500 calories, divided as follows: 10 per cent., 350 calories (87.5 grams) protein and of the remaining 3150 calories, one-third, 1050 calories (262.5 grams), in carbohydrate and 2100 calories (233.3 grams), fat.

The ulcer patient's optimal diet should be continued for a year or more. It is not necessary for him to weigh and measure his food as may be done if he is in a hospital, but during his fourth week of treatment, he may be taught to estimate his diet so that he may order a meal in a restaurant or at home, containing approximately the proper amounts of proteins, carbohydrates and fats. It is not possible even when food is weighed and measured to tell exactly the number of calories of protein, carbohydrates and fats that some items contain, and since the organs concerned with digestion and nutrition normally have a wide range in

function an error of 300 or 400 calories a day is not of moment.

The ulcer patient should weigh himself once a week. If he gains above his normal weight, cream and butter should be reduced; or if he is losing on his calculated optimal diet, a condition of infrequent occurrence, the diet may be increased until he is up to standard weight.

The Role of Vitamins In The Treatment Of Gastric And Duodenal Ulcers.

The problem of lowered resistance, or of the conditions in the stomach or duodenum that predispose to infection, resulting in ulcer, is yet to be solved. However, some of the facts brought out in recent studies on nutrition, particularly with reference to diets deficient in vitamins, if applied to the eating habits of the American people may suggest methods of preventing and curing gastric and duodenal ulcers.

About fifteen years ago, McCollum, Simons and Parsons[9] expressed the opinion that the role of food in the etiology of many diseases "involves increased susceptibility to infection, due to lowered resistance caused by faulty diet." Other workers in nutrition have come to the same conclusion.

McCarrison[11], a British army surgeon, stationed in a remote region of the Himalaya Mountains, made some observations and experiments which may give the key to an inportant predisposing cause of ulcers of the stomach and duodenum, as well as to other abdominal diseases due to infections. McCarrison was impressed with the fact that the Himalayans rarely had ulcers of the stomach and duodenum, or other abdominal infections, though they lived under the most unsanitary conditions. He stated that in nine years practice among the Himalayans, during which time he performed more than 3,600 major operations, he "did not see a case of ulcer, or appendicitis, or of mucous colitis." In his investigations as to the cause of this remarkable freedom from abdominal diseases among a primitive people, he became convinced that the use of "natural foods, i. e., milk, eggs, grains, fruits and leafy vegetables," protected them against infection.

McCarrison Evperiments. McCarrison's classic experiments seem to prove that foods of low vitamin value if used over long periods of time predispose to infections of the gastro-intestinal tract. He placed thirty-six healthy monkeys in two separate cages; twelve were fed on "natural" foods, i. e., milk, eggs, grains, fruits and leafy vegetables, and all of them remained healthy and free from gastro-intestinal diseases. He fed twenty-four monkeys on a sterilized carbohydrate diet, i. e., autoclaved rice, and most of them developed gastro-intestinal lesions, three out of ten autopsies having shown gastric ulcers present.

McCarrison is of the opinion that vitamins B and C serve to protect the gastro-intestinal tract from infections, and that the deleterious effects of a deficiency of these vitamins is enhanced when the food is improperly balanced, particularly when associated with an excess of carbohydrates. He states that "impairment of the protective resources of the gastro-intestinal mucosa against infecting agents may be due to hemorrhagic infiltration, to atrophy of the lymphoid cells, and to imperfect production of gastro-intestinal juices. This impairment not only results in infections of the mucous membrane itself, but also permits of the passage into the blood stream of micro-organisms from the bowels."

McCarrison showed, by illustrations of sections of various parts of the gastro-intestinal tract, that all these changes occur in animals fed on diets poor in vitamins and with an excess of carbohydrates. He asserts that "gastric and duodenal ulcer, colitis, and failure of colonic function can be produced experimentally by means of feeding animals on faulty food." He does not state that the faulty diet is the only cause of these gastro-intestinal conditions, but believes that pathogenic organisms are contributing factors. As proof that a faulty diet lowers resistance to infections, McCarrison fed endameba histolytica to healthy monkeys and failed to infect any of them, while those on a deficient diet became readily infected when given endameba histolytica organisms.

The Relation of Faulty Diets to Ulcers and Other Abdominal Infections. If McCarrison is right in his conclusions that a diet deficient in vitamins B and C predisposes to gastro-intestinal infections, many sugar saturated, vitamin starved Americans, i. e., those who live largely on white flour bread, white potatoes, white rice, lean meats, sugar saturated coffee, and sugar laden desserts, with candy and soft drinks betweeen meals, would seem to be

susceptible to ulcer and other abdominal diseases in which infection plays a part. The fact that more than 25 per cent of the patients in the general hospitals in the United States are admitted because of abdominal infections, i. e., appendicitis, gall bladder disease, ulcers of the stomach and intestines, and colitis, may be accounted for partly by the unbalanced diet of the American people.

Even casual observations of the eating habits of people in cafes, hotels, dining cars and their homes will reveal the fact that the starches and sugars and lean meats which are devoid of vitamins make up a large proportion of the diet of all classes of people, and that fruits and vegetables and milk, which are rich in vitamins, are eaten but sparingly by the masses. It is true that as a result of the increased interest in diet, educated, well informed people are eating better balanced meals than ever before; but all students of nutrition agree that the great fault in the diet of the average American family is the excessive use of vitamin-free carbohydrates.

A study of the quality and amounts of foods consumed by patients before they developed symptoms of ulcer may throw some light on the question as to whether or not a high carbohydrate, poor vitamin diet predisposes to the disease. It likewise would be of interest to study the food habits of ulcer patients in whom there were recurrences following operation or dietetic and medical treatments.

Sugar Saturated, Vitamin Starved Americans. For a quarter of a century before McCarrison's classic experiments, and many years before Rosenow's work proving the relationship of infections to ulcer, Deeks contended that a high carbohydrate diet, particularly cane sugar products, was responsible for the increasing incidence of gastric and duodenal ulcers, but on the theory that the irritating effects of carbohydrate fermentation in the gastro-intestinal tract was the etiologic factor. Deeks has recently revised his views and feels that an added factor is the deficient vitamin diet of those who eat excessively of sugars and the foods of high starchy content that are now in common use.

Pari passu with the increase in ulcers of the stomach and duodenum and other abdominal infections has been the increased consumption of sugar, syrups, white flour, white corn meal, white rice, white potatoes and other devitalized, or devitaminized, carbohydrates. The statistics on sugar consumption are available and are significant. Our consumption of sugar has increased nearly 500 per cent in the last half century, from 23 pounds (10 Kg.) per capita in 1870 to 106 pounds (48 Kg.) in 1926. In other words, every man, woman and child in the United States eats on an average one-third pounds or about a teacupful, of sugar a day. Statistics on the increased consumption of other devitaminized carbohydrates were not available at the time this paper was prepared, but there can be no question of their increased use in the United States.

Concomitant with the unprecedented increase in the use of sugar and other devitaminized carbohydrates has been the passing, particularly in the rural districts, of the home garden, orchard, dairy and poultry yard, which were formerly important adjuncts of every farm and which supplied the protective foods required for perfect nutrition. When these facts are considered, sugar saturated, vitamin starved America presents a problem which should be considered seriously by the medical profession, and the laity should be given the facts regarding the dangers of an excessively high carbohydrate, low vitamin diet. It is only through an enlightened public opinion that the perverted appetitie of a nation can be corrected.

Vitamins A and B. The recent advances in the study of nutrition have brought out some amazing facts, which should be applied in the treatment of ulcer. Without going into a discussion of the proven facts regarding vitamins, it is an accepted conclusion by nutrition experts that vitamin A protects against respiratory and eye infections, and that it stimulates nutrition generally is well known.

It would, therefore, seem that in dieting an ulcer patient the general nutrition of the patient should be considered and that his diet should be rich in vitamin A. Milk fat (cream, milk and butter) is the best source of vitamin A; though the green vegetables which should make up an important part of an ulcer diet after the first week are rich in this protective food.

No one doubts that the anti-neuritic vitamin protects against beriberi; and McCollum thinks that there are hundreds of thousands of border-line cases of nervous maladies that occur because of a de-

ficiency of vitamin B in the diet of the average American. The neurotic element, as a predisposing factor in the etiology of ulcer, is accepted by many gastro-enterologists; and since vitamin B is essential for perfect nutrition of the nervous system, whole wheat bread, vegetables and fruits—important sources of vitamin B—should be part of the diet of ulcer patients.

Dr. Crile observed that removal of the adrenals in animals is frequently followed by the development of duodenal ulcer. It is known that in fatigue there is a deficiency of adrenal secretion and the injection of adrenalin relieves fatigue. McCarrison is of the opinion that vitamin B is necessary for proper adrenal function. There is also evidence suggesting that a deficiency of vitamins affects thyroid and other internal secretions. It therefore seems that since the general use of white flour, white meal, white rice, and white sugar, all devoid of vitamins, a deficiency of vitamin B in the average diet may have something to do with the apparent increase of duodenal ulcer during the last few decades.

Vitamin C Protects Against Abdominal Infections. The anti-scorbutic vitamin, C, seems not only to protect against the disease known as scurvy, but against infections of the entire gastro-intestinal tract. It, therefore, would seem advisable for the ulcer patient to have a diet rich in vitamin C, with the view not only of aiding in healing the ulcer but also to keep him, as nearly as possible, in a state of perfect nutrition to prevent recurrences of the infection.

Fruit and raw vegetables are the best sources of vitamin C and since it has been proven that they do not increase gastric acidity, there is no reason why strained tomato juice, or the strained juice of other uncooked fruit and vegetables should not be begun early in the diet of the ulcer patients. Patients always like orange juice, it rarely disagrees, and there is no reason why it may not be given on the first day of treatment of ulcer cases. The first six days we give one feeding at 9:00 P. M., of the same quantity, from one-half to three ounces, of orange juice, with the milk and cream mixture. After the first week, orange juice, three ounces, is given with breakfast; and three ounces strained tomato juice with evening meal. Since the heat necessary for canning tomatoes does not destroy vitamin C, if fresh tomatoes are not available the strained juice from canned tomatoes may be used. Some patients do not like tomato juice, but when told that it is both food and medicine, they take it and soon cultivate the taste for it. It is sometimes necessary to prove to the patient that the old idea of acid fruits and acid vegetables being incompatible with milk is a fallacy; but a few feedings of orange or tomato juice with meals made up largely of milk and cream, soon convince him that there is no such incompatibility.

Diet for the Average Ulcer Patient Weighing 154 Pounds (70 Kilograms). It is not possible to prepare a diet suitable for all ulcer patients; but it is practicable to construct daily menus for the average ulcer patient, which may be modified, if necessary, to meet the varying nutritional needs of the individual under treatment. We use the following simplified diet for the average medical ulcer case. It is so constructed that it may easily be changed, increasing or decreasing the amounts of carbohydrates, proteins and fats to meet the estimated requirements in any given case.

SIMPLIFIED DIET FOR MEDICAL ULCER PATIENT

First Day: Every hour from 7 A. M., to 7 P. M., one-half ounce of a mixture of one-third cream to two thirds milk (thirteen feedings). One-half ounce strained orange juice after milk at 7 A. M., 1 P. M., and 7 P. M.

Second Day: Every hour from 7 A. M., to 7 P. M., one ounce of a mixture of one-third cream and two-thirds milk. One ounce strained orange juice after milk at 7 A. M., 1 P. M., and 7 P. M.

Third Day: Every hour from 7 A. M., to 7 P. M., (inclusive), one and one-half ounce of a mixture of one-third cream and two-thirds milk. One and one-half ounces strained orange juice after milk at 7 A. M., 1 P. M., and 7 P.M.

Fourth Day: Every hour from 7 A. M., to 7 P. M., (inclusive), two ounces of a mixture of one-third cream and two-thirds milk. Two ounces of strained orange juice after milk at 7 A. M., 1 P. M., and 7 P. M.

Fifth Day: Every hour from 7 A. M., to 7. P. M., (inclusive), two and one-half ounces of one-third cream and two-thirds milk. Two ounces strained orange juice after 7 A. M., 1 P. M. and 7 P. M., milk.

Sixth Day: Every hour from 7 A. M., to 7. P. M., (inclusive), three ounces of a mixture of one-third cream and two-thirds milk. Two ounces strained orange juice after milk at 7 A. M., 1 P. M., and 7 P. M.

Seventh to Tenth Days. 7 A. M., Breakfast: 3 ounces strained orange juice, 6 tablespoonsful strained oatmeal or one shredded wheat biscuit, with 2 ounces cream—no sugar. 1 slice dry toast of whole wheat bread. One soft boiled egg. 1 pat butter. 3 ounces milk.

10 and 11 A. M.: 1 ounce of cream and 2 ounces of milk.

12 M., Dinner: 3 ounces strained tomato juice. 1

tablespoonful scraped beef, slightly broiled. 1 slice whole wheat bread, toasted. 1 pat butter. 3 ounces milk.

2, 3, 4 and 5 P. M.: 1 ounce of cream and 1 ounce of milk.

6 P. M., Supper: 3 ounces strained orange juice. 6 tablespoonsful strained oatmeal or one shredded wheat biscuit toasted, or dry toast of whole wheat bread. 1 pat butter. 2 ounces cream. 1 soft boiled egg. 3 ounces milk.

Eleventh to Fourteenth Days. 7 A. M., Breakfast: 3 ounces strained orange juice. 6 tablespoonfuls strained oatmeal or one shredded wheat biscuit. 3 ounces cream. 1 soft boiled egg. 1 slice whole wheat bread toasted. 1 pat butter. 3 ounces milk.

9 and 11 A. M.: 3 ounces milk and 1 ounce cream.

12 M., Dinner: 3 ounces strained tomato juice. 2 large tablespoonsful scraped beef or minced breast of chicken. 1 slice whole wheat dry toast. 1 pat butter. 2 tablespoonsful ice cream, 3 ounces milk and 1 ounce cream.

3 and 5 P. M.: 3 ounces milk and 1 ounce cream.

6 P. M., Supper: 3 ounces orange juice (strained). 1 soft boiled egg. 1 slice whole wheat bread, toasted. 1 pat butter. 3 ounces milk—2 ounces cream.

9 P. M.: 3 ounces milk and 2 ounces cream.

Fifteenth to Twenty-first Day. 7 A. M., Breakfast: 3 ounces strained orange juice or strained grapefruit juice. 6 tablespoonsful strained oatmeal, cream of wheat or other smooth cereal—no sugar. 3 ounces cream. One soft poached or soft boiled egg. One slice toast, whole wheat bread, and two pats of butter.

10 A. M.: 5 ounces milk (half cream).

12 M., Dinner: 4 ounces strained tomato juice or strained vegetable soup. 4 rounded tablespoonsful scraped beef or minced breast of chicken. 4 rounded tablespoonsful tender green vegetables, as turnip greens, spinach or string beans (mashed through a sieve. 1 slice whole wheat bread toast; two pats butter. 1 rounded tablespoonful ice cream, cup custard, boiled custard or gelatine and cream.

3 P. M.: 5 ounces milk (half cream).

6 P. M., Supper: 4 ounces thick puree of peas or beans. 4 tablespoonsful tender green vegetables as turnip greens, spinach or string beans mashed through a sieve. 1 slice whole wheat bread toasted; two pats butter. 5 ounces milk. 3 ounces strained orange juice.

9 P. M.: 5 ounces milk (half cream).

Fourth, Fifth and Sixth Weeks. 8 A. M., Breakfast: 3 ounces strained orange juice. Six tablespoonsful strained oatmeal, or one shreaded wheat biscuit, or other cereal (no bran); three ounces cream—no sugar. One egg, soft boiled, poached or scrambled. One slice dry toast, whole wheat bread; two pats butter.

10 A. M.: 5 ounces of milk (half cream).

1 P. M., Dinner: 4 ounces strained tomato juice, clear broth or tomato broth, or strained vegetable soup. 4 to 6 rounded tablespoonsful scraped beef or minced chicken or lamb. 4 to 6 tablespoonsful turnip greens, spinach or string beans, or carrots, mashed through a sieve. One slice dry toast, whole wheat bread; two pats butter. 4 tablespoonsful ice cream, boiled custard or gelatine.

4 P. M.: 5 ounces milk (half cream).

6 P. M., Supper: 4 ounces thick puree of green or dried peas or butter beans. 4 to 6 tablespoonsful turnip greens, spinach or string beans or carrots, mashed through a sieve. One slice whole wheat bread toast; two pats butter. 4 ounces milk, 4 ounces strained orange juice.

9 P. M.: 5 ounces milk (half cream).

DIET AFTER SIX WEEKS

After six weeks the ulcer diet may be increased or decreased to keep the patient at his normal weight and in a perfect state of nutrition. With that end in view, while undergoing treatment he should be taught the simple facts regarding the vitamin values of food and the importance of continuing to eat a well balanced diet for the rest of his life. This does not mean food restriction, or what not to eat, as much as it does the necessity for taking a sufficient amount of food to keep up strength and endurance, as well as to eat enough of the protective foods to prevent a recurrence of the ulcer.

Especially should he be taught the dictum of McCollum's perfect nutritional day; i. e., that each normal person should drink from a pint to a quart of milk a day, and eat one raw fruit, one raw vegetable and two cooked leafy vegetables daily. In addition to that, he should eat a reasonable amount of meat once a day, one piece of bread, preferably made of whole wheat flour, or country ground corn meal, with liberal quantities of butter with each of three meals a day, and then a dessert of fresh fruits, cooked or uncooked, without sugar, or perhaps ice cream, sherbet or other light sugar dessert at one meal. This diet gives the patient with healed ulcer a wide variety of the most palatable foods known to man. He soon realizes that on such a diet he receives the greatest enjoyment from eating; he is comfortable and efficient, and he experiences the joy of living more than he did when he ate without a thought of whether or not the quality and quantity of foods he consumed met his daily nutritional needs.

Diet After Six Weeks. Breakfast: One fruit—Strained orange or grapefruit or cantaloupe juice. One cereal—small portion of thoroughly cooked oatmeal, or other cereal, no bran, or one shreaded wheat biscuit with one-half glass cream, no sugar. Eggs—one egg poached or soft boiled, or soft scrambled. Bread—one slice whole wheat bread toast, two or three pats butter; two tablespoonsful honey. Milk or cocoa—one glass milk or cup of hot cocoa.

Three Hours After Breakfast: 1 glass milk (one-third cream).

Dinner: Soup—strained chicken, celery, vegetable, barley soup, or strained tomato juice. Tender meats (small portion)—broiled, or baked, not fried; small portion of chicken, turkey, mutton, roast beef, bacon, thinly sliced boiled ham, or fish. Tender green and yellow vegetables (one variety of each)—large serving spinach, turnip greens, tender string beans, cook-

ed without much grease, carrots, squash, or young turnips. Butter or mayonnaise or olive oil and lemon juice may be used freely on vegetables after they have been cooked. Bread—one slice whole wheat bread or dry toast, or small piece of country ground corn meal bread, or one small corn muffin; two or three pats of butter. Milk—one glass milk or buttermilk (one-third cream). Desert—soft part of baked apple, thoroughly ripe banana or other fruit with cream, no sugar. Ice cream or gelatine, or sherbet, twice a week.

Three Hours After Dinner: 1 glass milk.

Supper: Soup—Puree (thick strained soup) of peas or beans, or oyster stew, no oysters. Raw vegetable salad—Crisp lettuce (trim out tough stems); peeled tomatoes (cut out rough centers); crisp cole slaw made of leafy part of tender cabbage (cut out the stems of the leaves). Olive oil and lemon juice or apple vinegar, or mayonnaise dressing may be used freely. Two crisp crackers. Cheese—four tablespoonsful home made or dairy made cottage or cream cheese, or the equivalent of two tablespoonsful of Swiss, Philadelphia, or yellow American cheese, never cooked. Bread—one slice wholewheat bread or dry toast, or six crackers. Two or three pats butter. Milk—one glass sweet milk, or buttermilk (one-third cream). Desert—strained orange juice, soft part baked apple, or very ripe banana and cream without sugar, or gelatine, or egg custard.

DIETARY MANAGEMENT OF PEPTIC ULCER PATIENTS BEFORE AND AFTER OPERATIONS

The problem of dieting the ulcer patient before and after operation is entirely different from that involved in the effort to heal gastric and duodenal ulcers medically; and the recognition of the pathologic physiology in the surgical patient, while applying the accepted principles of nutrition adapted to the needs of the individual under treatment, will reduce very materially the operative mortality of ulcer. Likewise, rational postoperative dietary management will prevent many recurrences of the original ulcer as well as safeguard against the dreaded jejunal or gastro-jejunal ulcers which follow only too frequently after gastroenterostomies.

The failure to get the ulcer patient in the best possible state of nutrition before operation and haphazard postoperative dieting account for the deaths from acidosis, shock and myocardial weakness of many asthenic individuals whose resistance has been lowered by years of malnutrition from pyloric stenosis with resulting gastrectasis. It also is true that the infections causing postoperative pneumonia, pleurisy, peritonitis and other complications following gastro-enterostomy and gastric resection may be prevented in many cases if the patient is properly prepared for the operation by a few days of dieting suited to his particular needs. Therefore, the surgeon should not consider a gastric operation as merely a mechanical procedure, but should recognize the nutritional problems involved, and when possible he should associate with him in the management of his ulcer patient a physician who has had the proper training and ample experience in dealing with such cases. It is still better for the surgical ulcer patient to be under the care of the physician and let him call in the surgeon to perform the operation when the patient has been properly prepared.

Nutritional Preparation of the Ulcer Patient Before Operation. There can be no routine diet and no fixed period of time required to prepare an ulcer patient for operation. Each case presents problems that have to be worked out to meet the nutritional requirements of the individual under treatment. The well-nourished ulcer patient, without gastric retention, and with a normal or high degree of free hydrochloric acid in his stomach contents after a test breakfast, requires very little or no preoperative preparation. For breakfast and dinner the day preceding operation he may have soft diet, with no meats and restriction of fats; and for supper, one or two crackers, or a piece of toast with jelly or honey, and a glass of skimmed milk or buttermilk. No butter, cream or other fats should be given to any patient for twenty-four hours before a gastric operation.

It is advisable for even the well-nourished patient to have at least 3000 c.c. of fluid during the twenty-four hours preceding a gastric operation; and the nurse in charge should be instructed to see that he gets that much water and other fluids. The fluid intake should be charted, recording the time and amounts of water, dextrose orangeade, dextrose lemonade, skimmed milk or buttermilk, or other fluid; and the surgeon should know that the patient has had approximately 3000 c.c. of fluids during the twenty-four hours before operation. No food, not even milk or strained orange juice, should be given the morning of the operation, and drinking water should be stopped at least four hours before the patient is carried to the operating room.

Preoperative Preparation of Ulcer Patients with Gastric Retention. Many ulcer patients do not come to operation until they have suffered for many years and repeated exacerbations of a very chronic condition, which disturbs nutrition, not only by absorption of the toxic substances

resulting from infection, but by actual starvation and dehydration because of weeks or months of gastric retention from pylorospasm, or actual pyloric obstruction. Dehydration and starvation predispose to post operative acidosis, alkalosis and infection; therefore the important indications in the preparation of the ulcer patient with gastric retention for operation is the free use of fluids and soluble carbohydrates.

The time required for the preparation for an elective operation on an ulcer patient with gastric retention cannot be fixed because it depends upon his state of nutrition and his vitality as to whether or not be can survive a serious abdominal operation. Not less than three or four days should be required in the fairly well-nourished patient with retention; and from one to three weeks are necessary in preparing the emaciated, anemic and dehydrated patient with gastric retention of over 50 per cent six hours after the barium meal.

Fluids and Soluble Carbohydrates Before Gastric Operations. Soluble carbohydrates may be given freely, but no protein foods should be given for twenty-four hours before operation in the ulcer patient with pyloric obstruction because water passes out of the stomach slowly during gastric digestion, and there may be retention of food and fluids in the stomach if milk or any other food containing protein is given. Fats are retained in the stomach even longer than proteins, and an excess of fats is a cause of acidosis. Therefore, no fats should be given to ulcer patients for at least twenty-four hours before operation.

Strained lemon juice, one ounce, to five ounces of a ten per cent dextrose, or glucose solution makes delicious lemonade; or strained orange juice, two ounces to four ounces of a ten per cent glucose or dextrose solution (orangeade) may be given in six ounce feedings every hour during the day, and every two or three hours at night if the patient is awake up to within six hours of the operation. This will give the patient from 250 to 300 grams (1,000 to 1,200 calories) of glucose and about three litres (six pints) of fluid in the twenty-four hours. If the dextrose or glucose solution cannot be retained by mouth, it should be given by hypodermoclysis in a five per cent dextrose or glucose solution, or intravenously in ten per cent solution in quantities of from 500 to 1000 c.c. every four to six hours. Intravenous dextrose or glucose solutions, properly prepared and properly given, often saves the lives of patients before and after gastric operations.

There are few hospitals in the United States that are properly equipped to prepare their own dextrose or glucose solutions, and most of the reactions following intravenous glucose solutions are due to faulty technic in its preparation. Therefore, it is best to use the ampules, prepared by reliable pharmaceutical houses, containing 50 grams of a 50 per cent solution of dextrose, the contents of which added to 200 c.c. of double distilled water will make a 10 per cent solution, or if added to 450 c.c. of distilled water will make a 5 per cent solution. If the patient is very weak not more than 500 c.c. of a 10 per cent solution should be employed, because there is danger of overloading a feeble heart. The intravenous dextrose solution should be given very slowly, requiring at least twenty minutes to introduce 500 c.c. of a ten per cent solution into the vein through an 18 guage needle.

The Futility of Giving Glucose or Dextrose Solutions by the Rectum. Dextrose or glucose solutions when given by the rectal route are of questionable value. Having observed flatulency and abdominal discomfort in a number of our operated ulcer cases that we thought were due to the use of glucose and water by the Murphy drip, we request the surgeons who operate for us to spare the rectum and colon except for plain water enemata. Recent studies seem to prove that very little, if any, dextrose or glucose is absorbed from solutions given by the rectum. From experimental and clinical studies by McNealy and Williams[11], Pressman[11], deTakats[13] and Schmidt and Cary[16], it seems that the use of glucose or dextrose solutions per rectum not only is futile, but may be actually harmful to the ulcer patient either before or after operation. Apparently American surgeons are discontinuing the use of proctoclyses of all kinds after gastric surgery, because as Strauss says they prefer to keep the intestines at absolute rest."

In a very timely article on the dangers of the surgeon's order to "push fluids," deTakats calls attention to "the fear of dehydration that has swept the country." deTakats thinks that 3,000 to 4,000 c.c. in twenty-four hours is as much fluid as any patient should have before or after any

operation; and that where there is myocardial damage, or kidney insufficiency or both, the fluid intake should be reduced very materially. The dehydrated, emaciated ulcer patient often has some cardiorenal damage and a weak heart may fail, and poorly functioning kidneys may allow water retention with the symptoms of what Rountree" calls "water intoxication" if an excessive amount of fluids is given.

Certainly the safest and the most accurate methods of administering dextrose solutions, Ringer's solution and saline solutions before or after operations are subcutaneously and intravenously. The rectum should not be disturbed except to give plain water enemata, not more than 1,000 c.c. by the Murphy drip method once or twice a day before and after operations. If the patient cannot take fluids by mouth and one feels that he must give fluids by the rectum before or after an operation, undoubtedly the safest, surest and most comfortable method is the use of from 200 to 300 c.c. of warm tap water by enemata, given slowly through a soft rubber catheter, every two or three hours.

Transfusions. Transfusions in preparing the undernourished, dehydrated, anemic ulcer patient for operation are of inestimable value. A transfusion of from 250 to 500 c.c. of blood provides the recipient with much needed nourishment, as well as other vital ingredients needed to carry him over the crisis of an operation. If the patient is very anemic and emaciated, one or more transfusions before and after operation may be a life-saving procedure. Undoubtedly transfusions are not employed as often as they are needed in the preparation of ulcer patients for operations.

Postoperative Dietary Management. In dieting after gastric operations the state of nutrition of the patient, the ulcer pathology, and gastric physiology should be considered. A distinguished surgeon is credited with saying that no patient who cannot survive a week's starvation should submit to a gastric operation. It is certainly true that the amount of food that can be given safely to a patient for the first week after gastric operation amounts to only a fraction of what he will metabolize during that time. He, therefore, must depend largely upon his own tissues (endogenous katabolism) for sustenance. The reserve glycogen in the liver, muscles and other tissues will be utilized first, then the fats will be burned and finally the proteins will be katabolized.

Subcutaneous and Intravenous Dextrose Therapy. One thousand c.c. of a five per cent glucose solution given by hypodermoclysis every eight hours after operation will give the patient 150 grams of glucose (600 calories) in twenty-four hours, sufficient carbohydrates to insure proper metabolism of the patient's endogenous fats, thus preventing acidosis. If the patient is vomiting or if he is very weak for the first two or three days after a gastric operation, it may be best to alternate with, or substitute for, the subcutaneous dextrose solutions 500 to 1,000 c.c. of a ten per cent dextrose solution intravenously.

Glucose or dextrose should not be given subcutaneously in over five per cent solution because of the pain produced by the more concentrated solutions. It may be given in ten per cent solution intravenously provided it is given very slowly.

The urine of the postoperative ulcer patient should be examined for sugar and diacetic acid once or twice a day, and blood sugar determinations should be made at least once a day in serious cases. If the carbohydrate tolerance is proved to be low and if there is diacetic acid in the urine, ten or twenty units of insulin given hypodermatically may save the patient from acidosis.

Water and Food by Mouth After Operations. Surgeons differ as to when water may be given by mouth after gastric operations. Since water passes out of the stomach in a few minutes when it contains no food, it would seem that after twenty-four hours no harm could be done by giving a few sips, one or two teaspoonsful, of either hot or cold water every one or two hours during the day, increasing it on the third and fourth days to two ounces, and to three ounces on the fifth day. Certainly the sooner the patient can get his daily quota of water by mouth, without distention of the stomach the better it is for him. The amount of water and other fluids retained should be recorded every day, reducing the subcutaneous and the intravenous use of dextrose solutions and the water per rectum as the patient can take more fluids by mouth.

A SIMPLIFIED POSTOPERATIVE ULCER DIET

It is not possible to construct a diet that may be used in every ulcer case that has been operated upon any more than a rou-

tine diet can be used with every ulcer patient treated medically, but we have prepared a diet that will meet the nutritional needs of the average postoperative patient while at the same time taking into consideration the ulcer pathology and gastric physiology involved. It may be modified to suit the indications in the individual case. Copies are given to the resident surgeon, or surgical interne, the nurses and the hospital dietitian with instructions to change the orders only with the approval of the surgeon or the associated physician.

First Twenty-four Hours: No food or water by mouth. Two hundred fifty c.c. plain warm water by slow enema every two hours. If the patient is vomiting, or in shock, or if there is diacetic acid in his urine, 1,000 c.c. of a five per cent dextrose solution is given subcutaneously every eight hours. Five hundred c.c. of a ten per cent dextrose solution intravenousiy may be substituted for the subcutaneous dextrose once or twice a day. Water per rectum should be regulated so that the patient does not get more than 3,000 to 4,000 c.c. of fluid a day.

Second Day: No food. Begin with one teaspoonful hot or ice water by mouth every hour. If no nausea, increase water to four teaspoonsful every hour, day and night when awake. Fluids and dextrose solutions subcutaneously, intravenously and per rectum as for first day.

Third Day: Two ounces (60 c.c.) cool water every two hours day and night when awake. Two ounces ten per cent glucose lemonade, or glucose orangeade, at 7:00 and 10:00 A. M., 1:00, 4:00, 7:00 and 11:00 P. M., and 4:00 A. M. May substitute two ounces (60 c.c.) weak strained tea for 7:00 and 10:00 A. M., and 1:00 P. M., glucose solution. If fluids are retained by mouth reduce the glucose or dextrose solutions by 500 c.c. and water per rectum by 500 to 1,000 c.c.

Fourth Day: Three ounces (90 c.c.) cool water every two hours when awake day and night, alternating with three ounces (90 c.c.) of weak tea with one teaspoonful sugar (without cream); or three ounces of a ten per cent glucose lemonade, or glucose orangeade, or three ounces of gingerale. Tea may produce wakefulness if given after 4:00 o'clock in the afternoon, particularly when given to one who does not ordinarily drink tea or coffee. The water by proctoclysis and glucose solutions by hypodermoclysis, or intravenously, may be discontinued on the fourth day if the patient can take fluids by mouth; but if nausea and vomiting persists, they should be be given as on the first day.

Fifth Day: Water and the ten per cent glucose lemonade, or glucose orangeade, or grape juice, or gingerale may be increased to four ounces and given every hour and a half, the water alternating with the tea, glucose solution, grape juice or gingerale every three hours.

Sixth Day: Water should be given freely between feedings, not over four ounces (120 c.c.) at a time. One egg albumen and three ounces of ten per cent glucose lemonage, or glucose orangeade, or four ounces gingerale every two hours from 8:00 A. M., to 7:00 P. M., and every three or four hours at night if awake.

Seventh to Tenth Days: 7:00 A. M., Breakfast. Three ounces strained orange juice. Four tablespoonsful of strained oatmeal gruel, with two ounces cream. One soft boiled egg. One slice white bread toast with crust trimmed off. 1 pat butter.

10:00 and 11:00 A. M. Five ounces (150 c.c.) dextrose orangeade, or five ounces gingerale.

12:00 M., Dinner. Three ounces (90 c.c.) puree of peas, beans or potatoes. One slice white bread toast. 1 pat butter. Two rounded tablespoonsful of plain jello or ice cream.

3:00 and 5:00 P. M. Five ounces strained tomato juice, or five ounces dextrose or glucose orangeade or five ounces gingerale.

6:00 P. M., Supper. Three ounces strained orange juice. Four tablespoonsful strained oatmeal—two ounces cream—no sugar. One soft poached egg on white bread toast. 1 pat butter.

9:00 P. M., and 1:00 and 2:00 A. M.—if awake. Five ounces strained tomato juice, or five ounces of dextrose or glucose orangeade, or five ounces gingerale.

Eleventh to Fourteenth Days: 7:00 A. M., Breakfast. Three ounces strained orange juice. Six tablespoonsful of strained oatmeal—with two ounces cream. One soft boiled egg. One slice white bread toast with crust trimmed off, and one pat butter.

10:00 and 11:00 A. M. Five ounces (150 c.c.) dextrose orangeade, or five ounces gingerale.

1:00 P. M., Dinner. Five ounces strained tomato broth, or strained vegetable soup, or creamed soup. One slice white bread toast. 1 pat butter. Two rounded tablespoonsful plain jello or ice cream.

3:00 and 5:00 P. M. Five ounces strained tomato juice, or five ounces dextrose or glucose orangeade, or five ounces gingerale.

6:00 P. M., Supper. Three ounces strained orange juice. Six tablespoonsful strained oatmeal or cream of wheat—2 ounces of cream—no sugar. One soft poached egg on white bread toast and 1 pat of butter.

9:00 P. M., and 1:00 or 2:00 A. M., if awake. Five ounces strained tomato juice, or five ounces dextrose orangeade, or five ounces of gingerale.

If the ulcer patient is comfortable at the end of two weeks after the operation the diet as advised in the medical treatment may be given. It is just as important for the surgical ulcer patient to be dieted for at least a year after the operation as if he had been treated medically.

General Instructions to the Convalescent Ulcer Patient. The patient with ulcer while undergoing either medical or surgical treatment should be taught good eating habits. He should be shown the necessity of thorough mastication and told that the use of peppers, spices and condiments is an acquired and wholly unnecessary habit, which he should avoid. Hot biscuits, fresh rolls, and corn bread with tough crust, fried foods, pies, pastries, cakes and syrup should be eliminated from his dietary. Likewise, tough meats, green corn, strawberries and other berries, fruits with tough skins, and vegetables with woody fiber, such as old okra and asparagus, should be omitted. Coffee, tea, caffeine beverages, wines, beer, whiskey or other

alcoholic beverages, since they increase the gastric acidity, should not be used by the patient who has recovered from ulcer. Tobacco should also be interdicted.

It is not enough to tell the ulcer patient in general terms of the foods which he should eat or which he must not take; he should be given a diet list with specific instructions regarding the portions of the various dishes for each meal. The patient should also be taught that perfect digestion depends not only on having the proper quantity and quality of food but on many other factors, such as overwork, worry, fear, anger, grief and other emotional disturbances which may predispose to ulcer.

While the dietary management of the patient with ulcer is most important, diet alone is not sufficient in treating the disease. Medicines, as indicated, should be employed. It is likewise necessary to examine every organ in the body for possible complications, because not infrequently other diseases may be found.

It is especially important at the beginning of treatment to search for possible foci of infection in the nasal and accessory sinuses, the teeth, tonsils, gall-bladder, pancreas, appendix, colon recto-anal region the urostatic gland in the male and the genital organs of the female, and the extremities. Of course, if a possible focus of infection is found, it should receive appropriate treatment and be removed if possible.

There is no need for the patient who has been cured of ulcer to regard himself in any other way than as a normal person, but he should report at frequent intervals to his physician for advice regarding diet and other matters pertaining to personal hygiene.

SYSTEM OF HYGIENIC LIVING FOR THE HEALED ULCER PATIENT

The great majority of properly treated ulcer patients, whether medical or surgical, may look forward to becoming more efficient and to enjoying life more than they ever did before; provided they follow the same rules of personal hygiene that every man in health should observe. The ulcer patient perhaps will live longer than he would have had he not developed an ulcer, because if taught properly how to live, as the physician or surgeon has the opportunity of doing, he will take such good care of himself that he will be less susceptible to many of the infirmities of middle life, and old age will thus be deferred.

A copy of the following outline of a system of hygienic living with copies of simplified diet lists prepared to suit his particular needs is given to each of our ulcer patients before they are dismissed.

1. **The Proper Diet.**—Three medium meals a day at the same hours, and a glass of milk between meals for at least three months after treatment; then one raw fruit, one raw vegetable, two cooked tender green vegetables, a pint to a quart of milk every day for the rest of the ulcer patient's life. Meat and light desserts are allowed not more than once a day; and enough bread, potatoes, rice, butter and other simple foods to maintain the normal weight. Thorough mastication of food is very important.

2. **Work—Avoid Overwork.**—Six to eight hours of honest work a day for five days a week and three or four hours on Saturday.

3. **Sleep.**—Eight or nine hours in bed at night; and lie down, sleep if possible, for half an hour after the noon meal.

4. **Exercise.**—Fifteen minutes room exercises with windows wide open, before the morning bath. Follow the bath with massage (brisk rubbing with open hand) of the entire body until the skin is reddened. A walk of one or two miles in the open air and sunlight each day, or what is better, play golf two or three afternoons a week.

5. **Recreation.**—Eight hours for play includes morning exercises, bath, golf, or other outdoor exercises; time for eating at least one-half hour for each meal, perhaps an hour for dinner and no work or serious reading after six o'clock in the afternoon. Frequent week-end trips and an annual vacation of from two weeks to a month every year.

6. **Serenity.**—Worry, anger, grief, abnormal fears, or other emotional disturbances will break down resistence to infections. Therefore, the cured ulcer patient should cultivate serenity in all the relations of life.

7. **Eliminate the Toxins.**—Coffee, tea and the so-called cola drinks, which contain caffeine, a habit forming drug, should not be used by the ulcer patient, except occasionally as a stimulant after losing sleep; or as a drug for headache or shock. Tobacco and alcohol are predisposing causes of ulcer and are quite injurious to the ulcer patient. Therefore, he should never use them in any quantity, even after his ulcer has been pronounced cured.

8. **The Annual or Semi-annual Physical Examination.**—The ulcer patient should report to his physician once a month for six months, and then for the rest of his life have an annual or semi-annual physical examination, with particular reference to a possible return of the ulcer; or the possible development of a cancer of the stomach, which, if diagnosed early, can be cured by operation.

Each section of the above outline of hygienic living is discussed with the patient and he is shown how he can conform his daily habits to a regular systematic regimen. He is also impressed with the fact that his future health, efficiency and happiness depend largely upon his living

the simple hygienic life, and that he must do his part in preventing a recurrence of his ulcer.

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RENAL FUNCTION IN ARTERIAL HYPERTENSION

Laurence B. Ellis and Soma Weiss, Boston (Journal A. M. A., March 25, 1933), used the usual urinalysis, urea and creatinine clearance tests and concentration-dilution tests in a study of the renal function of twenty-four cases of arterial hypertension without clinical signs of cardiac or renal failure, and in eight cases of glomerulonephritis. In ten of the cases of hypertension the urea clearance test gave normal results; in nine there was a slight reduction, and in five it was markedly lowered. Creatinine clearance tests, in eighteen of the same cases, were normal in thirteen instances, slightly reduced in two, and markedly reduced in three. In general, the outcome of the urea and creatinine tests tended to give parallel results. In the twenty-two cases in which the concentration test was performed in the patients with hypertension, the maximum specific gravity of the urine was above 1.025 in six instances; ten times it fell between 1.020 and 1.025; and in the remaining six cases it fell below 1.020. In only two instances did the concentration test show a definite lowering without a marked reduction in the urea or creatinine clearance tests. For practical purposes, in most cases a carefully conducted concentration test is as sensitive an index of renal impairment as the urea and creatinine clearance tests. Marked albuminuria or hematuria was uncommon in hypertension unless great limitation of function was detectable by other tests. The authors analyzed the results of their study in accordance with the filtration-reabsorption theory of renal physiology and correlated them as far as possible with the histologic changes in the kidney, known to occur in hypertension. There was a trend for the degree of impairment of renal reserve to parallel the height of the arterial blood pressure, particularly the diastolic pressure. There was no correlation to be made between the degree of renal damage and the age of the patients, the symptoms or the known duration of the disease.

INVESTIGATIONS AND CONCLUSIONS OF COMMITTEE ON COSTS OF MEDICAL CARE

Lewellys F. Barker, Baltimore (Journal A. M. A., March 25, 1933), presents the recommendations of both the majority and the minority of the Committee on the Costs of Medical Care. The majority reports: (1) that medical service, both preventive and therapeutic, should be furnished largely by organized groups of physicians, dentists, nurses, pharmacists and others, organized preferably around a hospital, the service to include home, office and hospital care, with the maintenance of high standards and the development of preservation of a personal relation between physician and patient; (2) that all basic public health services be made available to the entire population according to the needs; (3) that the costs of medical care be placed on a group payment basis, through the use of insurance, of taxation or of both, though service on an individual fee basis for those who prefer it need not be precluded; (4) that the study, evaluation and coordination of medical service be considered important functions for every state and local community and that agencies for the exercise of these functions be formed; and (5) that certain much needed improvements in undergraduate and postgraduate medical instruction be made, that the social aspects of medical practice be given greater attention, and that the practice of the specialties be better controlled. The minority recommends: (1) that government competition in the practice of medicine be discontinued and that government activities be definitely restricted to certain special fields; (2) that government care of the indigent be expanded so as ultimately to relieve the medical profession of the burden; (3) that the recommendation of the majority with regard to the functions of each state and local community in the study, evaluation and coordination of medical service be approved; (4) that united attempts be made to restore the general practitioner to the central place in medical practice; (5) that the corporate practice of medicine, financed through intermediary agencies, be vigorously and persistenly opposed; (6) that methods that can rightly be fitted into our present institutions and agencies wihout interfering with the fundamentals of medical practice be given careful trial; and (7) that state or county medical societies develop plans for medical care.

THE JOURNAL
OF THE
Oklahoma State Medical Association

Issued Monthly at Muskogee, Oklahoma, under direction of the Council.

VOL. XXVI AUGUST, 1933. No. 8

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Entered at the Post Office at Muskogee, Oklahoma, as second-class mail matter July 28, 1912.

This is the official Journal of the Oklahoma State Medical Association. All communications should be addressed to The Journal of the Oklahoma State Medical Association, 525-526 Commercial National Bank Building, Muskogee, Oklahoma. $4.00 per year; 40c per copy.

The editorial department is not responsible for the opinions expressed in the original articles of contributors.

Reprints of original articles will be supplied at actual cost provided requests for them is attached to manuscripts or made in sufficient time before publication.

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Failure to receive The Journal should call for immediate notification of the editor, 810 Manhattan Building, Muskogee, Oklahoma.

Local news of possible interest to the medical profession, notes on removals, changes in address, births, deaths and weddings will be gratefully received.

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PRINTED BY HOFFMAN-SPEED PRINTING CO., MUSKOGEE

EDITORIAL

YOUR FELLOW-MAN

Certainly we physicians may do well in attempting to emulate the actions of the great men of the past. The writer has recently read a most entertaining volume "Jefferson and Hamilton," by Claude G. Bowers, a great man himself, writer, orator, diplomat and statesman, now holding the post of Ambassador to Spain.

In culling through the actions and lives of Hamilton and Jefferson, Bowers notes that in their political relations they were like "two cocks, constantly at each others throats," for they represented widely divergent political views at a time when our nation was in its infancy and in the process of construction. However, Jefferson was a kindly man. A visitor to Monticello, the beautiful home of Jefferson, noted two busts, one of Hamilton, the other of Jefferson, facing each other across the hall. His comment was "opposite in death as in life."

We would highly recommend this attitude upon the part of our profession. It is said that Jefferson always left an opening for reconciliation with old and valued friends with whom necessity had forced him into temporary divergence of opinion. Jefferson made as few enmities as possible, and later, at some propitious time attempted to, and was usually successful in closing what might have been a life-time breach of cordiality.

What a wonderful thing it would be if our profession would cultivate that attitude. Of course, among the fifteen hundred members we have, some men are hardly worth while paying attention to, but, in the main there is some good in the worst of us, and it is human to have enmities arise. What a wonderful thing it would be to see the small number of physicians in Oklahoma towns working in harmony; yet it is not uncommon to see two men, often good men, pass each other by without the courtesy of even speaking. Often this state of affairs has arisen over the veriest triviality, the "corpse" over which it has arisen sometimes not being worth the snap of one's finger. What a wonderful thing it would be, say to see, five men in a small town working in harmony, remembering that there is both good and bad in the most of us. Their work could be divided by mutual agreement, all doing "general practice," but each selecting some one of the specialties and devoting his spare time to that. Soon you would hear less of this "send the case to Mayo's," they, jointly would be able to handle it, and properly at home. Such a situation would build up a local clinic almost before the builders were aware of their work. It seems utterly silly to see an Oklahoma physician sending a case hundreds of miles away, when right across the street is a man competent to handle the case properly, at a saving of a long, tedious and costly trip, and the money could be kept at home. You may be sure that the physician sending such cases away never gets a penny in return, on the contrary the prestige of his local profession is lowered in the eyes of the community, and all the sender gets is sometimes a

nicely typewritten note of thanks for his trouble.

Build up your home fires and keep them burning. Let cooperation be your watchword, practice this in your daily dealings and soon the bread you cast upon the waters will be returned many fold to you. Get acquainted with your fellowman, stand by him, insist that he stand by you, and soon you will be rewarded by practical results.

DR. CHARLES ROBINSON HUME—A BIOGRAPHY

Dr. Charles Robinson Hume was born in Monroe County, State of New York, October, 1847. When a lad his parents removed to Southern Michigan. He attended the common schools of the neighborhood, and Oak Grove Academy at Medina, Michigan.

In 1871, he matriculated as a medical student in the University of Michigan, from which school he graduated in 1874. He began the practice of medicine in Perrysburg, Ohio, and in 1876, was married to Miss Annette Ross of that city.

In the winter of 1880-81, with his family, he removed to Caldwell, Sumner County, Kansas, on the border of the Cherokee Strip, then in Indian Territory, now Oklahoma.

In 1890, he was appointed resident physician in the U. S. Indian Service on the Kiowa and Comanche Reservation, holding the position for eleven years until this reservation was opened to white settlement.

In the organization of Caddo county he was appointed as first County Health Officer, holding that position until statehood seven years afterwards. He was the first president of the Caddo County Medical Society, and Secretary-Treasurer of the organization for twenty-two years following. He has been a member of the Oklahoma Territory and State Society since soon after its organization. In 1917, he was elected president of the Oklahoma State Medical Association, and represented the state society at the American Medical Society as delegate in 1918 at Chicago, where a thousand medical officers were in uniform, and the 1919 session at Atlantic City, just after the close of hostilities.

During the World war, Dr. Hume was a member of the Medical Advisory Board, District Fourteen, Oklahoma, and also a member of the Volunteer Service Corps authorized by the Council of National Defense.

Mrs. Annette Ross Hume passed away January 19, 1933, after more than fifty-six years of married life. She was active in the State Federation of Women's Clubs, and in religious work. She was a member of the Daughters of the American Revolution, Colonial Dames, and up until the time of her death, took active part in the religious and social functions in the State.

They had two sons, Dr. Raymond Robinson Hume of Minco, Oklahoma, and Judge Carlton Ross Hume of Anadarko, Oklahoma.

Dr. R. R. Hume spent a year in the Medical Service overseas, during the war, at which time he was a Captain.

Judge Hume was the first person to receive the M.A. degree from the University of Oklahoma, and his son Ross G. Hume, is in active practice in law in Oklahoma City.

Dr. Charles R. Hume has a great-grandson, Charles Robert Hume, which makes four generations of Humes in Oklahoma.

Editorial Notes—Personal and General

DR. L. W COTTON, Enid, has been appointed Health Superintendent of Garfield County.

DR. C. R. SILVERTHORNE, Woodward, who has been ill for several weeks, is reported improved.

DR. C. E. WILLIAMS, Woodward, has been reappointed to the County Excise Board of that city.

DR. AND MRS. A. H. McMAHON, Duncan, have returned from a visit to the Chicago World's fair.

DR. AND MRS. J. S. ROLLINS and children, have returned from Chicago, St. Louis, and a tour of the Ozarks.

DR. CLAUDE A. THOMPSON, Muskogee, who has been ill for the past several weeks, is reported improving.

DR. ROSS JACKSON has located in Mangum. Living in Texas for several years, he has returned to his old home in Mangum.

DR. JOHN D. CAMPBELL, formerly of Houston, Texas, has located in Duncan, and will be associated with Dr. B. H. Burnett.

DR. F. W. BOADWAY, Ardmore, recently made

his seventeenth annual visit to the Kiamichi Mountains on a fishing expedition.

DR. J. W. CRAIG, Miami, has returned from an extensive trip over the Northern States, including attendance at the American Medical Association.

ANADARKO'S City Superintendent of Health is not satisfied with the sanitary conditions of the city and has ordered a general cleanup of alleys, etc.

DR. G. S. BARGER, Purcell, accompanied by his family, returned from a trip to St. Louis, Missouri, where they attended the wedding of his son Blanchard.

DR. AND MRS. WALTER HARDY, Ardmore, attended the A. M. A Convention at Milwaukee, and stayed over for a few days at the Century of Progress Exposition, Chicago.

DR. R. M. SHEPARD, Tulsa, attended the National Tuberculosis Association in Toronto, Canada, June 26-30. Dr. Shepard invited the association to hold its 1935 meeting in Tulsa.

DR. L. J. MOORMAN, Oklahoma City, President of the local Tuberculosis Society, and Vice-President of the National Society, attended the society's convention held in Toronto, Canada, in June.

CARTER COUNTY, after years of litigation has had its rights restored to levy a tax for the support of the health unit, upheld by the Supreme Court. The unit is in charge of Dr. John L. Dorough.

DR. PAUL L. SCHROEDER, University of Illinois, Chicago, delivered a post-graduate lecture recently at El Reno on "Mental Hygiene of Infancy and Childhood." Dr. T. M. Aderhold, El Reno, acted as clinic chairman.

DR. WM. J. WHITAKER, Pryor, announces the near completion of his hospital. He will have as an assistant Dr. R. H. Walker, a young surgeon now in San Francisco, who formerly was connected with the McBride Reconstruction Hospital in Oklahoma City.

DR. H. K. SPEED, Jr., Sayre, a new arrival in the field of Oklahoma Medicine, is a son of Dr. H. K. Speed. He graduated from the University of Oklahoma, passed the State Board and will be associated with his father for a short time after which he will take up interneship.

TULSA BOARD OF EDUCATION will discontinue its School Heaith Clinic according to press dispatches. Dr. W. A. Walker, Director of the Board, states that preparations had been made for seventy tonsillectomys and more than four hundred forty dental cases during July.

GARVIN COUNTY MEDICAL SOCIETY met July 19th, at Pauls Valley for its monthly meeting. Dr. L. P. Smith, Elmore City, read a paper on "Some Obscure Aliment of Infancy and Childhood"; Dr. Geo. N. Bilby, State Health Commissioner, Oklahoma City, delivered an address.

ST. JOHN'S HOSPITAL, Tulsa, has offered to take over the care of county patients, promising a reduction of approximately $30,000. The Board of Governors and staff of the hospital stated that in their opinion the county work should be divided between the two larger hospitals, St. John's and Morningside.

OKLAHOMA COUNTY COMMISSIONERS AND CITY MANAGER made a sensible and long step forward when they merged the county and city medical services into a coordinated system. The city will continue its dispensary, dispensary physicians being empowered to send needy cases to the County Hospital. A night physician will be maintained throughout the year. Dr. Basil Hayes was appointed County Physician and Dr. George Borecky was appointed field physician.

CARTER COUNTY MEDICAL SOCIETY met in regular session at the First Methodist Church, Monday night, July 10th. A banquet was served by the Ladies of the Missionary Society. Following the banquet was an interesting program on public health subjects:

Address of Welcome—Dr. A. Y. Easterwood, Ardmore, City Health Officer.

"Milk"—Dr. M. M. Miller, Washington, D. C., United States Public Health Service.

"Mental Hygiene"—Dr. J. L. Day, Norman, Physician Central State Hospital.

"Camp Hygiene"—Lt. Baine, Turner Falls, Director C. C. C.

"Milk versus Beer"—Dr. Weith, Pastor of the First Presbyterian Church, Ardmore.

The meeting was open to the public for attendance. There were thirty-two physicians, osteopaths and densists and about fifty laymen present.

DR. E. S. LAIN, Oklahoma City, has received notice that he has been awarded a Class I award for Medical Research work on "Electro-galvanic Lesions of the Oral Cavity Produced by Artificial Dentures." Notice of this award may be found under the report of the Committee of Awards, Journal of the A. M. A., July, 8, 1933.

OTTAWA COUNTY ITEMS

Dr. J. M. Lanning, formerly of Picher, but later of Shawnee, has located in Miami. He is associated with Dr. J. M. Kitchen, formerly of Picher. They have offices in The Miner's Exchange building.

Dr. F. C. Evans of Tulsa, recently located at Afton, for the practice of his profession.

Dr. A. M. Cooter, the dean of Ottawa County's Medicos lost his wife Tuesday, July 26th, after a day's illness of cardiac asthma.

Dr. J. M. Dawson of Afton, a pioneer Oklahoma physician is critically ill at the home of his daughter at Afton. He is suffering from a general break down.

Dr. J. W. Craig of Miami, spent most of the month of June vacationing at Detroit, Toronto, Buffalo, and Chicago, where he attended the Century of Progress, for a week. He reports that the medical displays are very complete and interesting.

Dr. A. M. Cooter of Miami, left Thursday for a month's stay in Denver, Colorado.

Dr. Geo. A. DeTar and wife of Miami, spent a ten days' vacation in Colorado in forepart of July.

Dr. C. V. Powell, Government Physician of the

Seneca Indian School at Wyandotte, is taking his vacation, and his position is being filled by Dr. Goodwin, temporarily.

Dr. W. B. Smith of Miami, was appointed County Physician for Ottawa County by the County Commissioners at their July meeting, at a salary of $150.00, per month.

Dr. J. B. Hampton of Commerce, is taking a month's vacation in Texas.

DR. LAMBERT KUNTZ

Dr. Lambert Kuntz, 75 year old physician of Perry, died June 25th, of cerebral hemorrhage.

He was born in Berkenseld, Germany, in 1858, moving with his family to Sweet Springs, Missouri, at the age of ten.

He received his preliminary education at Sweet Springs, Missouri, and his medical education at Vanderbilt University.

Dr. Kuntz has been in active practice in Perry for the past twenty-five years.

He is survived by one daughter and two sons.

REPORT OF THE DELEGATES TO THE A. M. A. MEETING IN MILWAUKEE

At ten o'clock Monday morning, June 12th, the House of Delegates was called to order for the 84th annual session at the headquarters hotel in Milwaukee, with all three Oklahoma delegates present.

Since the last meeting, Dr. Albert E. Bulson, Fort Wayne, Indiana, Vice Speaker of the House of Delegates, had died, and Dr. Warnshuis appointed the Secretary, Dr. Olin West, to act temporarily as Vice Speaker, who took the chair while the Speaker gave his address. The Speaker pointed out that the medical profession was confronted by conditions such as they have never encountered before, which may necessitate revision of attitude and intensified activity with increased alertness as how best to enhance the vital interests of the public and the profession. Reference committees were advised to secure details concerning association affairs before they made their recommendations and all delegates were urged to investigate the sources of information before introducing any resolutions or motions dealing with association policies. It seemed as if the number of deaths of members of the House of Delegates during the past year exceeded that of any year during the history of your delegates and after paying our respects with a moment of silent prayer, the chairman announced the Reference Committees which were to function on all matters introduced during the session. Oklahoma had the honor of being placed on one of these important committees. The speaker then introduced our friend and neighbor, Dr. Edward H. Cary, President of the Association, who gave one of the most thorough talks on organized medicine and the things that the association has been doing that we have ever heard.

Dr. Cary spent eleven months of the year that he was president away from his home in Dallas, on association business and traveled 100,000 miles and was the association's official representative in 80 different engagements. We are safe in saying that Dr. Cary, gave more of his time to the office than any other president of the association. One of the principal points brought out by Dr. Cary was the contention of the profession that we should have the right in prescribing alcoholic liquors, which has been recognized recently. Dr. Cary went into the question of hospitalization of Veterans very thoroughly and seemed to think that the government should not build any more hospitals to take care of the veterans whose disabilities were not service connected and that seems to be the policy of the administration at Washington, at this time. The government has at present 41,000 beds in Veterans' Hospitals which should be sufficient, but if not, it would be much cheaper to hospitalize their excess patients in private institutions as the expense per patient of maintaining a Government Hospital is greater than a privately operated one. Dr. Cary mentioned the fact that the physician should not be interested in his success alone but should promote and advance the prevention of disease, teach the public how they can ward off illness and instruct them as to the foods most applicable for the different seasons of the year. He spoke of the value of the American Medical Association Journal. It was necessary to draw on the reserve to the extent of $76,000 to carry on the last year as it has in the past. The magazine Hygeia, which now goes into practically all physicians offices and many private homes, has reached the stage where it is self-supporting and will produce a revenue eventually. After Dr. Cary paid his respects to the prominent deceased members of the association, the President-elect Dr. Dean Lewis of Baltimore, was introduced.

Dr. Lewis made a short snappy talk which showed that the affairs of the association are to be in good hands and he will carry on where Dr. Cary left off. He said that the aim of the Medical Schools today is the desire to graduate the highest type of student who should be admitted to practice without examinations before State Boards.

Dr. Rudolph Matas, vice-president, was introduced. Dr. Matas said that the office of vice-president had been regarded as a sinecure and thought it was only conferred upon him has honorary but that many duties were passed on to him, and that he had spent a busy year. He said that he had spent most of the year in praying and getting all the help that he could from the churches for the preservation of the life of the President so that he would not be called upon to serve in that capacity in case anything happened to the president. Dr. Matas is one of the old school and probably no other physician has more loyal friends than Dr. Matas. We expected to have the pleasure of voting for him as president-elect but on account of the large amount of work shouldered on the president and his advancing years, his name was not placed as a nominee.

The reports of the officers was routine and have been published in the Journal in full, so anyone who is interested, has access to it and we will not bore you with any report in which you are not particularly interested.

After the reports of the officers, numerous resolutions were introduced. One, in which we were all interested, was the one introduced by Dr. Kopetzky of New York: That the Council of Medical Education and Health be authorized to deal with the listing of specialists in the future editions of the Medical Directory. This was referred to the Committee on Medical Education, who recommended its adoption, which was done at the Tuesday meeting. Another important resolution was relative to the election of delegates from State Societies, the gist of which was

that the term of delegates should run from January 1st, to December 31st, so that Dr. West would know in advance the name of all delegates elected for each session, but as it is now, some state societies have their elections only a short time before the annual American Medical Association meeting and the time is so short that the secretary does not have them in time to send to the speaker so he can have them available for appointing the reference committees. The newspapers were all set to get a lot of dope on birth control as Dr. Plass of Iowa, introduced a resolution endorsing same, but it was tabled and no doubt we will hear from it again next year. Dr. Mundt of Illinois, introduced the following resolution which is so practical that we will quote the entire resolution, "Resolved, that it is the opinion of the House of Delegates that the physicians on staffs of hospitals approved for interne training by the Council on Medical Education and Hospitals should be limited to members in good standing of their local county medical societies and that the House of Delegates request the Council of Medical Education and Hospitals to take this under advisement." This resolution was referred to the Council and will be reported and acted upon at the next session in Cleveland, Ohio. Dr. Bedell of New York, introduced a resolution, the gist of which, is the limiting of the number of medical graduates, as there is an over production of physicians as well as commodities in the United States. This resolution was referred to the Council of Medical Education and Hospitals, who are investigating the matter.

The House of Delegates met in Executive Session Tuesday afternoon, after which we adjourned until 1:00 P. M., Thursday, at which time the final reports were made by the committees and we proceeded with the annual election which resulted in the election of Dr. Walter L. Bierring, Des Moines, Iowa, as President-elect; Dr. John H. Musser, New Orleans, Louisiana, Vice-President; Dr. Olin West was re-elected Secretary, unanimously; Dr. Herman Kretschmer was elected to the office of Treasurer, which had been occupied for several years by Dr. Austin Hayden of Chicago; for the fourteenth time, Dr. F. C. Warnshuis was elected Speaker of the House of Delegates; Dr. Nathan B. Van Etten was elected Vice-Speaker; Dr. Austin A. Hayden of Chicago, was elected to serve as Trustee for five years, to take the place of J. H. Walsh of Chicago; Dr. C. B. Wright, Minneapolis, was elected Trustee for five years in place of Dr. Mitchell, Nebraska, who died a few days before the annual meeting. After a spirited contest, Cleveland, Ohio, was decided upon for the 1934 meeting and seemed to be the most practical place for us to meet as it is centrally located and has ample facilities as regarding hotels and meeting places.

Oklahoma was fairly well represented, for thirty-three of our members were present, which was more than any other state located as far from Milwaukee as we are. Thursday night, as is the usual custom, was given over to the President's Reception and Ball which was not as formal as usual as many linen suits were mingled with evening clothes.

Your representatives have no regrets that we voted for Milwaukee as we were royally entertained. I believe that the minor details of the meeting were arranged more carefully than at any place we have ever met but we anticipate as good a meeting in Cleveland in 1934.

DR. HORACE REED,
DR. McCLAIN ROGERS,
DR. W. ALBERT COOK.

R. B. DAVIS COMPANY COCOMALT

An alarming result of the economic depression through which we are passing is the tremendous increase in mal-nutrition among school children. A recent survey of 130,000 school children in 16 states showed that 21 per cent were underweight by 10 per cent or more.

One way in which school and medical authorities are meeting this grave problem—combating this ever-increasing menace—is by serving Cocomalt in milk to the youngsters at lunch time. Every glass is equal in food energy nourishment to almost two glasses of milk alone; and as a result the children very quickly show signs of mental and physical improvement. Wherever possible parents have been asked to cooperate by serving Cocomalt in milk at home. Children love this delicious chocolate flavor food-drink and drink far more of it than they would of milk alone. Very gratifying gains in weight and energy have been reported.

Cocomalt contains a rich supply of Sunshine vitamin D and is accepted by the American Medical Association Committee on Foods.

PABLUM—MEAD'S PRE-COOKED CEREAL

Mead Johnson & Co., are now marketing Mead's Cereal in dried pre-cooked form, ready to serve, under the name of Pablum. This product combines all of the outstanding mineral and vitamin advantages of Mead's Cereal with great ease of preparation.

All the mother has to do to prepare Pablum is to measure the prescribed amount directly into the baby's cereal bowl and add previously boiled milk, water, or milk-and-water, stirring with a fork. It may be served hot or cold and for older children and adults cream, salt and sugar may be added as desired.

Mothers will cooperate with physicians better in the feeding of their babies because Pablum is so easy to prepare. Please send for samples to Mead Johnson & Co., Evansville, Ind.

TUBERCULOUS FISTULA IN ANO

Because of a traditional belief that anal fistulas should not be operated on, they are often neglected in tuberculous patients. Clement L. Martin, Chicago (Journal A. M. A., July 15, 1933), proved 56 per cent of anal fistulas in fifty-five patients having pulmonary tuberculosis to be tuberculous; 72 per cent of twenty cases in 1932 were proved tuberculous, by more detailed investigation. That something more than 72 per cent are tuberculous is probable. Of all pulmonary tuberculosis cases at the Municipal Tuberculosis Sanitarium, 7 per cent presented anal fistulas. The diagnosis of tuberculosis in fistula of the anus and rectum in patients with pulmonary tuberculosis depends entirely on the histopathologic examination because the gross, the clinical and the bacteriologic results are indefinite or of no value. In 87 per cent of the patients with pulmonary tuberculosis on whom anal fistulectomy was performed, healing was complete; the remaining 13 per cent were far advanced cases; despite this fact, healing was progressing in 10.4 per cent of these up to the time of death. This does not confirm the claim that these wounds do not heal satisfactorily. In the tuberculous patient, a perianal abscess should be incised as soon as it is diagnosed. Drainage of the infected area must be maintained. If a sinus or fistula persists, it should be operated on as soon as the general condition permits.

ABSTRACTS • REVIEWS • COMMENTS AND CORRESPONDENCE

SURGERY AND GYNECOLOGY

Abstracts, Reviews and Comments from LeRoy Long Clinic
714 Medical Arts Bldg., Oklahoma City

The Interrelationship Between Ovarian Follicle Cysts, Hyperplasia of the Endometrium, and Fibromyomata. J. Thornwell Witherspoon, M.D., New Orleans, Louisana. Surgery, Gynecology and Obstetrics, June, 1933, Volume LVI, Number 6. Page 1026.

This article is an attempt to prove an association between ovarian follicle cysts and hyperplasia of the endometrium in the causation of histogenesis of fibromyomata uteri. The author reviews the various theories of etiology from a pathologic standpoint. He discusses the influence of heredity and race and the significance of sterility often associated with fibroids. He then presents the data which is quite conclusive to show the relation of hyperplasia of the endometrium to follicle cysts as a causative feature.

He then presents 26 cases who had previously been operated upon with a definite diagnosis of hyperplasia of the endometrium who subsequently required hysterectomy for fibroids. Follicle cysts were present in all and there was hyperplasia of the endometrium.

He then presents 83 cases on whom hysterectomy was done for fibroids showing a large predominance of follicle cysts and hyperplastic endometrium.

He then presents the third group of 41 cases of fibroids and hyperplasia of the endometrium, but the evidence is indirect since the ovaries were not removed for microscopic study, but were merely described at operation.

His summary of the article follows:

1. The various theories pertaining to the histogenesis and etiology of fibromyomata of the uterus are discussed.

2. Evidence is offered to show that the unopposed action of estrin, in the absence of the corpus luteum influence, is the cause of hyperplasia of the endometrium.

3. An hypothesis is advanced to suggest that the unopposed action of estrin on the myometrium, if prolonged sufficiently, would result in fibromyomatous growths.

4. Twenty-six cases of hyperplasia of the endometrium, in which operation was done and diagnosis as such made from microscopic study, and in which a second operation was performed for fibromyomata after an approximate interval of four years and four months, are analyzed.

5. One hundred and twenty-four cases of fibromyomata, diagnosed microscopically, are offered with their associated ovarian and endometrial findings, as presenting added evidence in support of a cause and effect relationship between ovarian follicle cystic formation, hyperplasia of the endometrium, and fibromyomata of the uterus.

6. An hypothetical conclusion is advanced on the uterus results: (1) in immediate endometrial changes, characterized by hyperplasia, and (2) in more latent myometrial pathology in the nature of fibromyomatous growths.

Comment: That the growth of uterine fibroids depends upon the ovarian function has long been established. As a matter of fact, one of the early operative procedures for the treatment of fibroids was the removal of both ovaries. From clinical experience and the careful reading of current literature, one is particularly impreessed with the fact that fibroids are very rarely present without either ovarian cysts or a clinical history which would signify their presence at some time during the menstrual life of the patient.

These theoretical discussions would at first glance have very little place in such correspondence as is attempted here, but upon the other hand the very common occurrence of the uterine fibroid, and the therefore constant demand for decisions to be made in its treatment, compel us to understand as much as possible about its etiology and growth so that we may not only properly treat it from the standpoint of the tumor, but from the standpoint of the patient as well.

Indeed, it may be possible in a not distant day to establish prophylaxis against the occurrence of fibroid tumor.

—Wendell Long.

Rupture of the Graafian Follicle, the Corpus Luteum and Small Follicle or Lutein Cysts Simulating Appendicitis. Joe Vincent Meigs, M.D., and W. Fenn Hoyt, M.D., Boston, Mass. American Journal of Obstetrics and Gynecology. Volume XXV, Number 4, April, 1933.

These authors have reported 25 patients with rupture or bleeding from the ovary who have been operated upon at the Massachusetts General Hospital. In many of these cases they feel that operation could have been avoided and that in making a diagnosis of appendicitis in young women a rupture of the ovary should be considered. "Rest in bed and careful observation might prevent unnecessary operation for mild acute appendicitis."

"Sudden onset of pain, low temperature, slightly elevated pulse, and low white count out of proportion to the pain are findings suggestive of this lesion."

"In reporting these cases and from the conclusions drawn, the authors feel a responsibility, for careless observation and unnecessary delay might endanger the life of a woman with acute appendicitis.

Comment: Rupture or bleeding from ovarian cysts is not at all an uncommon cause for pain which simulates very closely appendicitis. I should add to the facts which the authors bring forth a very careful menstrual history as very important, particularly in relation to the time of the pain in the menstrual cycle.

It must not be forgotten, however, that in case of

any grave doubt, removal of the appendix is always the safest procedure from the patient's standpoint.

—Wendell Long.

Tubular Adenoma (Arrhenoblastoma) of the Ovary. By Frank Spielman, M.D., New York. American Journal of Obstetrics and Gynecology, April, 1933. Vol. XXV, No. 4, Page 517.

This is a case report of an arrhenoblastoma of the ovary, interesting principally because of its rarity and its striking clinical features. There have been only 24 previous cases reported. The striking clinical features are masculinization of the female in the secondary sex characteristics as evidenced by hypertrichosis, deepening of the voice, coarsening of the features, increase in size of the clitoris, amenorrhea, loss of breast tissue and other changes. In this case the patient had been studied at Mt. Sinai Hospital where no palpable tumors were felt in the pelvis, but later died at another New York hospital from another illness and large tumor of ovary was discovered at autopsy.

The most significant work on these tumors has been done my Meyer, who divided them into three groups. First, the tumors have a regular and typical tubular microscopic arrangement. Second, a group showing an irregular or atypical arrangement of the tubules. Third, intermediate group showing both the typical and atypical features.

The etiology is discussed and attention is called to the fact that masculinization of the female is not exclusively associated with this tumor. "Ovarian cystadenomas, teratomas, carcinoma, as well as adrenal and pineal tumors may also produce the changes."

This author believes that normal studies offer the greatest hope for future enlightment. In types of abnormality described here, with or without tumor, these studies certainly should be made.

—Wendell Long.

Organotherapy of Mastodynia. Alexander C. Gabrielianza, M.D., Chicago, Ill. American Journal of Obstetrics and Gynecology, April, 1933, Vol. XXV, No. 4.

This author has presented a small number of cases of mastodynia (painful breasts) treated by ovarian residue and by the administration of female sex hormone. Of eight cases only one patient received no relief from the pain.

The cases are reported and the theories of etiology are discussed. The conclusion is drawn that the cause of mastodynia lies probably in over activity of the corpus luteum and that the primary cause of it is in the hyper-function of the anterior pituitary gland.

Comment: We have seen a number of cases of mastodynia who have been relieved by the administration of folliculin, which theoretically is the substance which should cause relief. However, it must be pointed out that painful breasts are very seldom seen as the single abnormality. They are ordinarily associated with other symptoms and with other evidences of glandular dyscrasia. Therefore, careful study of each case in relation to menstrual history, headache, dysmenorrhea, sterility, secondary sex characteristics, etc., are essential before determining the character of the therapy.

There is also enthusiasm now for the expectant treatment of small breast tumors on the basis of endocrine therapy. It cannot be pointed out too strongly that all definite breast tumors have malignant potentialities, and regardless of the age of the patient they should be removed and miscroscopic investigation carried out. Only in this way may women be prevented from unnecessary risk due to malignancy degeneration.

—Wendell Long.

Arrhenoblastoma of the Ovary. J. M. Taylor, M.D., S. J. Wolfermann, M.D., and Fred Krock, M.D., Fort Smith, Arkansas. Surgery, Gynecology and Obstetrics, June, 1933, Volume LVI, No. 6, Page 1040.

These authors are presenting a second case of arrhenoblastoma which they say is the twenty-seventh in the literature. In this particular case the patient was a young woman of eighteen who had begun to menstruate normally, then had amenorrhea and the masculinization symptoms, followed by removal of the tumor and return of menstruation with return to female secondary sex characteristics. The pathology and etiology are here also reviewed.

Comment: These tumors, though rare, are of importance, primarily, because they signify the tremendous potentialities of the ovary, secondarily, because they may be of importance in determining the hormonal function of ovarian tissue; and thirdly, because the clinical recognition offers such definite and striking benefit to the patient.

—Wendell Long.

EYE, EAR, NOSE and THROAT

Edited by Marvin D. Henley, M.D.
911 Medical Arts Bldg., Tulsa

Ophthalmology in the Bible and in the Talmud. Benjamin L. Gordon, M.D., Atlantic City. Archives of Ophthalmology, Vol. IX, No. V, May, 1933.

The object of this treatise is to introduce in a short space the art of ophthalmology as practiced by the Hebrews of the Biblical and Talmudic days, and not a general discussion of medicine as practiced in that era.

It is not easy to definitely establish the standing of medicine among the ancient Hebrews because many errors were made in re-writing manuscripts and verifying the Aramaic names of the ocular diseases. Also the Talmud covered approximately 650 years and was the work of nearly 4,000 students.

In that remote period ophthalmology was limited to the diseases of the external eye since the ophthalmoscope was not invented until in 1845.

Those well versed in Talmudic teachings tell us the Hebrew word for eye is ayin, meaning a well or spring, since it was thought that the source of tears was located in that organ.

The eyelids are called shmuros (watchers) because the lashes protect the eyes against foreign matter.

According to the Talmud the eyeball is made up of the following coats: ocular conjunctiva, sclera, cornea, choroid, retina, iris, and lenticular capsule.

Ishon is the name given the pupil. Translated Ishon means little men. Babas-Ayin means the baby of the eye, hence the tiny image seen when looking into the eye. The iris (Kesheth) is located at the junction of the black and white coats and is named Sira.

Ophthalmia covering all known congestive dis-

turbances and blindness from any cause were the two classes into which ancient ophthalmology was separated.

The Talmud describes numerous diseases both of the eyelids and the eyeball. Those of the lids are: (1) Jabeleth, a hard wart beneath the bulging skin. (2) Dildul, a tumor on the edge of the lid. (3) Blepharospasm, blinking eyes. (4) Ptosis, drooping eyelids. (5) Blepharitis ciliaris, supposedly caused from much weeping. (6) Trachoma or ophthalmia granulosa, caused by uncleanliness, improper diet, and the many sand storms. Before the exodus of the Hebrews in 3400 B. C., this disease was described under the name of "dripping eyes." (7) Ophthalmia neonatorum. (8) Zugdus, one eye weaker than the other. (9) Ankyloblepharon, adherence of the eyelids. (10) Phthyriasis palpebrarum (existing today in Palestine and Egypt among children), is black nits (pediculosis pubis) covering the eyelashes, causing redness and itching of the lids.

The various diseases of the eyeball as described in the Talmud are: (1) Dak, meaning macula, an opacity of the cornea which may be moveable or fastened tightly. (2) Chvarvar, a leukoma differing from dak in the denseness of the opacity. (3) Barkith (translated gray-star), Interstitial Keratitis, frequently mistaken for cararact. (4) Liphluph, pus in the anterior chamber. (5) Thebalul, coloboma of the iris and sclera. (6) Mayim (water) hakebuim was the Hebrew name for cataract. In Arabic it was called al-ma anmasil fel-ain, the flow of the water of the eye. Cataract was supposedly caused by a liquid running down from the head and lodging between the iris and the lens. (7) Mayim-Yerkuim (sea green star), glaucoma ophthalmia, arthritica, described a greenish discoloration of the pupil. It was also mistaken for a cataract and was the cause of much of the blindness of that time. (8) Enab, staphyloma of the cornea receives its name because it is a grape-shaped tumor, sometimes dark or gray but generally white in color. (9) Parasitic ophthalmia, ophthalmomyosis, was most unusual and caused by animal parasites entering the eye through the nose or lacrimal canal, going to the brain and causing damage to the eye. (10) Merida, exophthalmos, bulging of the eye. (11) Ukla, phthsis bulbi, a shrinking eyeball.

In Biblical times priests afflicted with "dripping eyes" or impaired vision were considered unfit for Holy office.

It is interesting to note the various treatments for some of the previously mentioned diseases. The bark of nut and fig trees, shavings of hides, application of verdigris with onion, rubbing the Milesian wool and cauterization followed by copper oxide were the favorite remedies for trachoma. Abstinence from water and thirst producing food was prescribed for cataract and crying was a good omen, since the flow of tears would eliminate the cause of the disease. The cure for staphyloma of the cornea was Tignah, a herb soaked in honey or a grape of corresponding color and dimension applied to the lesion. Another popular herb was Cohol. This was rubbed on the lid and was said to have aided the eyesight up to the age of 40, but after this age it was of no benefit. Later (25-50 A. D.), the white of an egg was used after operative methods were discussed. The galls of reptiles, animals, fish, and blood of a bat were used in corneal opacities. Common drugs in use were antimony, copper, iron, and lead. The leaves of Koriander, Busbarta and of Gargira were used on inflamed lids. Spittle is mentioned in the Bible several times as a cure of blindness and the Talmud prefers the spittle of the first born. Collyrium made up in liquid, semi-solid, and powder was another remedy used by antiquity. In Palestine this was purchased at public markets. Pliny records that a cataplasm collyrium was made up of soaked bread, wine, milk, or white of an egg added to the regular ingredients.

Spectacles at first were worn only by priests and elderly scholars afflicted with eye strain.

Talmud discusses artificial gold eyes. Preuss however believes that the gold eyes found in Egyptian mummies were put in place after death.

Comments: From this most interesting and splendidly written article by Gordon I have taken only those cases which are comparable with the diseases and cures of today. This is only a short resume of a fifteen to twenty thousand word monograph with a lengthy bibliography entailing infinite care in its composition.

Cavernous Sinus Thrombosis Following Submucus Resection. By Noah D. Fabricant, Chicago. Archives of Otolaryngology. Vol. XLII, No. V, May, 1933.

Fabricant reports the case of a male, age 33, who was examined and a submucus resection performed three months from the time of the original and only examination. At the time of the examination the findings were negative except for a markedly deviated septum. The regular routine of a submucus resection was completed without any particular difficulty. The nasal cavity was packed lightly with petrolatum strips for twenty-four hours. Two days after the operation he was discharged from the hospital. Two days later he returned complaining of an excruciating headache, which persisted the next day. He was re-admitted to the hospital where for the next three days his condition was mildly septic. A differential blood count showed ninety per cent polymorphonuclears. The operative field appeared clean and apparently free of infection. The temperature was never high, the maximum being 101 degrees F. A roentgenogram showed only slightly increased density over the right maxillary sinus. Negative findings were reported after numerous consultations. Oedema of both eyelids was noticed on the eighth day after the operation. Following this development protrusion of both eyes occurred in a period of a few hours. There was a slight tenderness over both juglar veins. He became increasingly lethargic and expired the following morning.

Turner and Reynolds in a summary covering twenty-two instances of septic cavernous sinus thrombosis, state that the pathways of infection were as follows: "In thirteen cases, infection reached the sinus entirely by way of its afferent of efferent venous channels from the face, pharynx, air cavities and the cleft of the middle ear, in eight, the preliminary stage of the infective process was direct extension through the bone, followed by further spread to the sinus along the osseous veins; in the latter group, the primary focus was in the frontal, ethmoidal or sphenoidal air sinuses. In the remaining instance the infection passed to the blood sinus from the sphenoidal air sinus entirely by direct extension of the inflammation through the bone and dura mater."

Although there was no positive findings at autopsy the author concludes that the infection most likely occurred by direct extension by continuity of tissue from the sphenoidal air sinus into the cavernous sinus. He points out the importance of taking a careful history and doing a thorough examination before any

operation since a history of swimming and cold was later obtained from the family.

Other grave complications mentioned of this operative procedure are: generalized septicemia, meningitis, hemorrhage, abscess of the brain and erysipelas.

Schwannoma of the Larynx. By Harris H. Vail, M.D., Cincinnati. Annals of Otology, Rhinology and Laryngology. Vol. XLII, No. 2.

Theodor Schwann, a German anatomist, of the early part of the nineteenth century first recorded his observations of a tumor formed by the elements of the delicate protoplasmic envelope enclosing every internodal segment of the adult medullated nerve fiber.

Dysphagia, dyspnoea, and dysphonia were the prominent symptoms of Vail's patient, age 50. These had increased in severity over a period of three years.

After a tracheotomy had been performed a bilobular tumor over two inches in diameter was removed from its pedunculated point of attachment which was the right aryepiglottic fold just above the arytenoid. Ether anaesthesia and a Lynch-Killian suspension apparatus were used. This was accomplished after a failure under ether anaesthesia without a tracheotomy.

The intraoral route is here used successfully without the sequela of a vocal cord paralysis which sometimes occurs after the use of the external route which as a rule is the method chosen for removal of all large laryngeal tumors.

Literature shows three similar cases, two diagnosed as neurinoma and one as fibroma, where the external route was used at operation. Two of these showed a subsequent vocal cord paralysis of the side from which the tumor was removed.

Nasal Obstruction in Children Due to Septal Abnormalities: What Shall We Do For Them? Dr. Wiliam Wesley Carter, New York City. The Laryngoscope Vol. XLIII, No. V. May, 1933.

The author here discusses a problem which is constantly recurring to the rhinologist. It is easy to explain deviated septums in active male adults who have participated in athletics of all kinds while in college, but how account for them in females who have always led sedentary and protected lives? The author attributes these to unobserved falls or blows during childhood, seemingly so slight as to be without significance at the time. The normal infant has a somewhat flattened nose. Vertical pressure on the septum elevates it into the more prominent adult organ. A slight dislocation of the septum in a child generally becomes more noticeable as time goes on, even changing the facial expression until we have a saddle back deformity or a flattened nose due to the fact that the nasal bridge has not been lifted by the septum.

Carter states that 75 per cent of the Caucasian adults have deviated septums. Negroes and Mongolians are seldom so afflicted because they have characteristic broad flat noses.

Trauma, producing a hematoma with subsequent abscess is the most frequent cause of nasal obstruction and deformity. Early recognition of hematoma and free drainage to prevent abscess formation and destruction of nasal cartilage is stressed.

If there is obstruction following traumatism sufficient to prevent proper aeration, operative procedure should be instituted, or the health and the proper development of the child will be impaired Preservation of the structural framework is of paramount importance in all corrective operative work of this type.

Comments: V. P. Blair suggests that the volume of nasal plastic surgery would be greatly reduced if the proper care were to be given at the time of the injury in infancy and early childhood. I think the percentage of deviated septums in Caucasians as given above is very conservative. Rarely do I find a nasal septum which is perfectly straight.

Filariasis Oculi. By K. Koman, Pillai Nayer, and A. Kandaswami. British J. Ophth., September, 1932.

The patient, an adult male, of middle age, lives in a community where filariasis is prevalent. For five days he noted an object floating in front of his left eye. Examination revealed in the vitreous just in front of the macula a thread-like wriggling worm, coiling itself and moving briskly. As accurately as could be determined it was about 2.5 mm. by .5 mm. After fourteen days of observation it disappeared. Five days later it reappeared in the anterior chamber, motility unimpaired. Subsequent observation of three days was terminated by opening the anterior chamber with a keratone. Despite elaborate precautions the worm was lost.

Micro-filaria (bancrofti) was repeatedly demonstrated in nightly specimens taken from the peripheral blood stream.

TUBERCULOSIS

Edited By

L. J. Moorman, M.D.
304 Osler Bldg., Oklahoma City

Types of Tuberculosis Lesions Found in the Chest of Students of Nursing and Medicine. By J. Arthur Myers. American Review of Tuberculosis, July, 1933.

This paper is written with a view of discussing development of tuberculosis in students of medicine and nursing and to give a description of the conditions which are now looked upon as leading to the development of such pulmonary lesions. A plea is made that more careful studies be made in this connection which will eventually lead to as near complete protection for students as possible.

The observations are made on students in the School of Medicine and the School of Nursing at the University of Minnesota. The students are divided into three groups: Group 1, cases concerning which almost nothing was known as regards tuberculosis until symptoms appeared: Group 2, cases about which little, but not enough, was known until symptoms appeared or the disease was detected by periodic examinations: Group 3, cases about which much was known when the disease made its appearance.

Group 1, included those students who came in for examination of their own accord because of symptoms of disease. There had been no tuberculin testing; no periodic examinations or X-ray films previous to the symptoms. In this group, only a few were fortunate enough to have such dramatic symptoms as pulmonary hemorrhage or pleurisy with effusion to arrest attention, while the majority presented minimal, or moderately advanced disease at the time of examination.

In the second group are included those about whom

some definite information was available before lesions appeared in the lungs but due to the intervening period, such previous information was not regarded as of much help. Several cases under this group are cited. A typical one is as follows:

A hospital intern, age 25, had been positive to tuberculin tests ever since he entered the School of Medicine. Films of chest made October, 1930, and June, 1931, showed no evidence of disease. In November, 1931, there was slight infiltration in the left second interspace with a questionable small infiltration in the first right interspace. The fact that the tuberculin test had been positive throughout his medical course was very helpful. The tuberculous lesions appeared later, so that, in the opinion of the author, they could be caused only by the reinfection or adult type of tuberculosis.

In the third group are those cases concerning which sufficient recent information was at hand to justify somewhat definite conclusions about the development of tuberculosis and the type of disease present. This group of cases was negative to tuberculin on admission to schools of nursing and medicine but periodic tuberculin testing subsequently revealed positive tests.

In this third group the series of cases presented are very much the same in that they were negative reactors to tuberculin on admission to the school but became positive reactors following definite exposure, either by nursing tuberculous patients or by service on tuberculous wards. In some of these cases the site of the primary infection, which gave rise to the allergic state and therefore a positive tuberculin test, was never definitely decided upon. In others, the infection progressed to a point of showing minimal symptoms or X-ray evidence.

The author points out from these illustrations that the adult tolerates well the first-infection type of tuberculosis. The author further concludes that the young adult who has been protected against tubercle bacilli and first becomes infected in adult life is in no greater danger of developing a rapidly destructive type of tuberculosis than the child. He also states that the so-called childhood infection is not necessary to protect the adult against destructive and fatal tuberculosis, but is always a fore-runner of this disease.

Because of the chronicity of the disease, the lesions found in groups 1 and 2; that is minimal, moderately advanced or advanced; could have been detected months, or even years, before the symptoms appeared if the authorities of the schools had been on the alert. That is, by demanding that tuberculosis be sought in the chest of every student entering the school of nursing and medicine, and that tuberculin tests and X-ray films be properly and adequately employed throughout every year the student remains in school.

In summarizing, the following are a few of the points brought forward by the author:

1. The incidence of tuberculosis among students and recent graduates of schools of nursing and medicine is so high that the disease may be looked upon as a serious menace to professional health-workers.

2. Nearly all of the cases examined by this author in the Minnesota School from 1920 to 1928 were in group 1, consequently these students had lost their best chance of ultimate recovery and had persisted as spreaders of tubercle bacilli.

3. Since 1928 tuberculin tests have been administered routinely to students entering the University of Minnesota. Since that time fewer cases have been found in group 1 and a good many have fallen into group 2. The number of cases with moderately and far advanced disease has definitely decreased. Almost no advanced cases are seen except on entrance examinations.

4. The positive tuberculin reaction is the first manifestation of invasion by tubercle bacilli. It precedes X-ray evidence by weeks and months, and in many cases it remains the sole evidence of tubercle formation.

5. In no cases in which first-infection with tubercle bacilla has occurred in adult life has any serious illness accompanied it. Therefore, the development of rapidly progressive and highly destructive disease (galloping consumption) in adults who have not been previously infected is apparently a myth.

6. A positive tuberculin reaction, indicating allergy resulting from first-infection type of tuberculosis, is a definite liability, since the destructive forms of tuberculosis do not ordinarily develop in nature in its absence. Therefore, the longer it can be postponed, the better.

The Effects of Thoracoplasty on the Heart. By Olga S. Hansen and Henry W. Maly. American Review of Tuberculosis, February, 1933.

Comments on studies by these authors is given.

It has been shown in this series, that the intrathoracic pathological involvement incident to thoracoplasty almost invariably (in 87 per cent) displaces the heart more or less to one side or the other, but more frequently toward the unaffected side. The electrocardiograms also show a high incidence of postoperative change (63 per cent, including the slight ones), but these changes are neither consistent nor predictable, being in agreement with the X-ray findings in only a third of the cases. It would seem impossible to predict the probable electrocardiographic change from a study of the X-rays, or conversely to guess the type of X-ray findings from looking at the electrocardiograms.

It is probable that the electrical axis may be influenced by rotation of the heart on its longitudinal axis by fibrotic tissue affecting at times the base and at times the apex anteriorly or posteriorly, as has been shown experimental by Meek and Wilson and others.

It is probable that other factors such as bed rest, toxemia, and weight changes may affect the form of the electrocardiogram, since the control patients who had no gross mechanical changes were also variable in their complexes.

There has been no evidence of disturbance in conduction or of myocardial damage in electrocardiograms. Autopsy has shown no abnormalities in heart-weight nor more evidence of myocardial degeneration than is found in other patients dying of tuberculosis. Some of the changes in Q. R. S amplitude probably represents changes in muscle-tone associated with reduction in toxemia and increase in exercise and would appear regardless of the mechanics of collapse. The changes found in the electrocardiogram are probably due to extrinsic factors and bear no relationship to the conditions of the heart muscle.

Marital Tuberculosis. By M. Davidson. Brit. J. Tuberc., April, 1932.

In contrast to the older authorities, who found a 4

per cent incidence of tuberculosis in married partners of tuberculosis patients, Ward has recently compiled figures which show 58 per cent of definite tuberculosis and 10 per cent of suspects among 156 married partners. Verco has shown that, in respect to conjugal infection, women appear to be three or four times as susceptible as men.

Since all individuals in civilized countries are infected with the tubercle bacillus and rendered allergic in the first few years of life an immunity is obtained which protects most of them from ordinary exposure. Married persons are no exception to this rule, but if one of the partners has advanced disease the risk of massive doses of tubercle bacilli and consequent exogenous reinfection in the consort are increased.

If precautions are observed there does not appear to be more risk to the married partner than to any one else in frequent contact with tuberculosis patients.

DERMATOLOGY, X-RAY AND RADIUM THERAPY

Edited by William E. Eastland, M.D.
Lain-Roland Clinic, M. A. Bldg, Okla. City

Radium in Medical Use in the United States. R. R. Sayers, M.D., Radiology, April, 1933.

This history of radio-active minerals is reviewed briefly, showing that Professor Henri Becquerel first found that uranium salts produced photographic impressions through opaque substances. He assigned to Marie Shloddowska (later Madame Curie) the task of working out the nature of these rays.

It is interesting to know that the first commercial radium was produced from the mines of Joachimsthu, Bohemia. In the United States the Bureau of Mines became active in radium production in 1912, but it was 1914, when they united with the National Radium Institute, when the product was actually put on the market in Denver, Colorado. 8.5 grams had been produced when the plant closed in January, 1917.

At this time there are only two countries that are producing radium to any significant degree, namely, the Belgian Congo at Chinkolobwe, Katanga, and Czechoslovakia at Jackymov. The annual yield for the two countries totals about sixty-three and a half grams, ranging in price from sixty to seventy dollars a milligram.

Other countries yield radium in the respective order: United States, Canada, Russia, Portugal, Madagascar, England and Australia.

In the United States radium was first produced in 1913, whereas, the last production was in 1926, yielding a total of 202.523 grams. In order to determine the amount of radium in medical use in the United States, a questionnaire was sent out to all hospitals listed in the 1931 Directory of the American Medical Association, which information was requested as to how much radium they had on hand, what type of an applicator it was in, and what particular salt of radium, etc. It was estimated from the reports that 80,000 patients are treated annually; 287 hospitals and clinics reported a total of 85,800.26 milligrams of radium, and 128 of the 287 each had 75 milligrams of radium or more; 414 physicians reported they have 33,286.93 milligrams and 171 of the 414 each had 75 milligrams or more; nine laboratories and companies report 5,545.42 milligrams and 5 of the 9 each had 75 milligrams or more. Five states reported no radium in hospitals, and no radium is owned in the state of Wyoming. Individuals, companies, and hospitals estimate they need 117.4 grams more in addition to 124.7 grams already owned.

Since cancer has increased to second place as the cause of death, it is very evident that much more radium is needed than is now owned.

Dosage of Skin Therapy Expressed in International Roentgen Units. Joseph Jordan Eller, M.D., and Arthur Mutscheller, Ph. D., Archives of Dermatology and Syphilology, Vol. 27, No 4, April, 1933.

The authors in this article publish the results obtained by a group of different dermatologists who have been working on the problem of standardizing X-ray dosage for skin therapy. Heretofore, the unit has been dependent upon obtaining the slightest degree of erythema on the skin with certain factors, or the smallest dose that produces a temporary alopecia of the scalp. It has been found that on an average it requires 340 roentgens (i. e., international roentgens) to obtain the clinical signs above mentioned. This information is very valuable in standardizing X-ray dosage.

The Trichophytin Test (Report of 350 cases). Emanuel Muskatblit, M.D., and William Director, Archives of Dermatology and Syphilology, Vol. 27, No. 5, May, 1933.

In view of the widespread fungus infection commonly known as "athlete's foot" and its multiple ramifications, more knowledge is being sought by general practitioners in regard to correctly diagnosing this disease. The trichophytin test has been in use for a number of years, and this article deals with 350 cases tested by the authors. Their summary and conclusions are quoted:

Summary

1. A method of preparation of a polyvalent fungus extract containing endoproducts and exoproducts of epidermophyton, trichophyton and microsporon is described.

2. Three hundred and fifty patients were tested intradermally, of whom 300 clinically were myocotic and 50 were not mycotic.

3. Nonspecific reactions were observed in 4 per cent of nonmycotic cases.

4. The trichophytin test gave a positive reaction in 72.3 per cent of cases proved mycotic by the laboratory.

5. Clinical diagnosis was more frequently corroborated by the test than by the laboratory examination; for example, in clinically mycotic cases the test was positive in 60.3 per cent, while laboratory examination was positive in only 39.7 per cent.

6. Patients with highly inflammatory lesions gave a higher percentage of positive tests; however a positive reaction was found in many cases with limited and slightly inflammatory lesions.

7. The test was positive in cases in which the infection was due to various fungi of human and animal types, particularly in those caused by epidermophyton interdigitale.

8. Monilia infections of the skin also gave a positive reaction but less frequently than cases due to

filamentous fungi (trichophyton, epidermophyton and microsporon).

Conclusions

1. The intradermal test with fungus extracts,, while not absolutely specific, is of considerable value in the diagnosis of fungus infections of the skin due to filamentous as well as to yeastlike fungi.

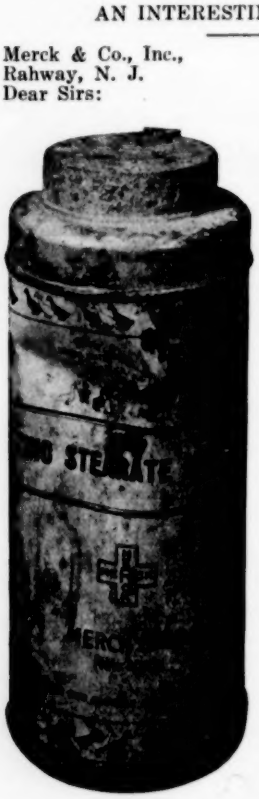
2. Common fungi such as trichophyton violaceum, microsporon audouini and epidermophyton interdigitale yield an effacacious preparation when a mixed polyvalent extract is made of freshly isolated strains.

3. General allergic hypersensitiveness of the skin develops not only in deep inflammatory mycoses but also in many cases of superficial type.

4. Epidermophyton interdigitale, the most common causative agent of dermatophytosis in this country, is apt to cause a considerable degree of cutaneous allergy."

AN INTERESTING LETTER

Merck & Co., Inc.,
Rahway, N. J.
Dear Sirs:



I was cleaning out an old well yesterday that hadn't been used, cleaned or lived around for over five years, and what do you think I found? One of your small cans of Zinc Stearate Merck Powder in the bottom.

I picked it up, saw the pictures of all the ducks and read all the printing on the can. When I read—it sheds water like a duck—that caused me to investigate and what do you think I found when I removed the top? A can about half full of powder and the remainder of the can filled with water. I poured the water off and let the powder remain in the can. The powder was just as dry and dusty as any powder could be.

I think your powder is a little better than you advertised it; you said it would shed water like a duck. Well I believe a duck would leak a little if it would stay in water for five years without coming out. I am almost sure the can of powder had been in the well for at least five years, if not longer.

Your truly,

JOSEPH MURPHY,
West Louisville, Ky.

THE DOCTOR AND POLITICS

Are the members of the medical profession aware of their entire responsibility to the community in which they live? Is the Oklahoma Medical Association aware of the responsibility which it has in protecting the medical profession in Oklahoma?

Throughout history it will be found that the physician and the position which he occupies in the community and the state within which he lives characterizes to some extent the degree of civilization and the culture and protection by which he is surrounded. By the word protection, I do not mean the keeping of the body from harm or the restraint of banks or loan companies from the foreclosure of notes and mortgages, or the preservation of a moral or social standing. But I do mean the preservation of the medical man's possibility by laws, that will guarantee him an equal right with any of the other business or professional men, to collect their fees.

Medical service has a definite market value. The benefits which are accomplished through personal administration of scientific principles cannot be fully compensated for by the dollar. The honest man pays you your fee and extends to you his undying gratitude for your service. Laws are not needed for this type of individual but on the other hand, there is a class of our citizenship which feel that it is a mental and physical accomplishment to accept of you your service and then refuse to pay. The merchant can recover his goods, the laborer can establish a lien on property, the lawyer can and does have ample time to draw up a binding contract; but the medical man acts in an emergency and if he attempts to acertain who will be responsible for the debt, he is classed among the money grabbers and advertised from mouth to mouth among the laity as a Doctor who places the dollar in advance of his patient.

Medical service is a commodity that is listed among the economic necessities of society in general, of industry and of the individual. Therefore, it is right that society, industry, and the individual pay the Doctor in proportion to the time he uses while rendering professional service. It is no more incumbent that the physician dispose of his time gratis than it is upon the business man to dispose of his wares free.

There should be uniform and consistent laws in the State of Oklahoma, and the citizenship of the State should be taught to recognize that the Doctor is a necessity to the community. Laws should be constituted whereby the medical profession will have equal rights with other professional or business enterprises to collect that which justly belongs to them.

I do not wish to insinuate that the people who are too poor to pay for medical attention should be neglected. It is not right that a doctor or a group of doctors in a community be forced by public sentiment to carry the burden of attending the people who are not financially able to recompense the medical man for his service. If a man disobeys the law and he is brought to the bars of justice, the court appoints and the county pays a lawyer to represent him. If the individual is kept in jail, food, and if necessary, raiment and medical care is provided for him. If this man's wife or children become ill, public sentiment is extended to her, the doctor makes his daily trips

or the necessary medical attention is rendered and in the larger majority of cases the doctor receives no compensation, not even a paltry sum from the county. If this family does not have funds or raiment, the county provides the same, or if not provided by the county, first one grocery store then another and another and finally by some philanthropic organization. They may not receive any compensation but the responsibility is divided among many, even this principle is wrong. The burden of caring or providing for the sick and poor should be based upon advalorem taxation and each individual should pay their prorated share, thus relieving the burden from the shoulder of the medical profession. At this point enters the title of the paper, "The Doctor and Politics."

The perfect physician will have to be a genius to begin with, unless he knows something in addition to doctoring, he must fall short of the ideal. The majority of medical men are public minded but they have forgotten to exercise it or in some cases, failed to recognize their responsibility in moulding the public policies in their community, city and state. Therefore, it behooves the medical man who is qualified by right of education, reasoning, thought and intimate knowledge of the needs of the people, in the community, to fill various public offices. If the medical man himself does not care to sacrifice time and energy for his community, he should at least pick out some public spirited citizen to whom he can express his opinions and ideas, and with whom he can exert sufficient influence to help mould the public policies. Extend to this man the influence which you yourself have, and see that he is elected.

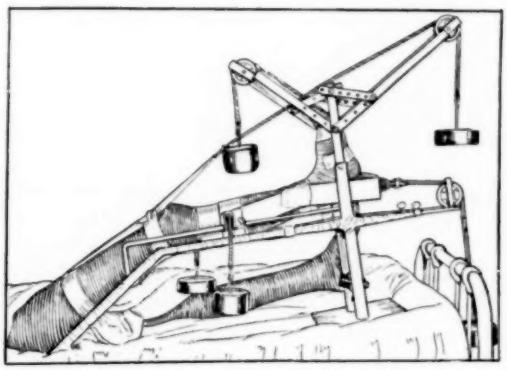
There is no profession or group of men in the State of Oklahoma who can yield such a powerful political influence as the Oklahoma State Medical Association. This influence is built up from the doctor in the community, to the county society, then to the district organization and finally to the state association. This powerful organization composed of educated public minded citizens lies dormant and allows less powerful political machines to elect men to public offices to make our laws and another group of men to execute them.

I have just recently finished my first experience as a State Senator of Oklahoma. While there I felt the powerful influence of lobbyists of various organizations. Everyone fights to create new laws or maintain those already in effect which protects their business or profession. The insurance companies, both local and foreign, spent many dollars in various forms of entertainment to forward their selfish interest where only a few thousands of dollars would be saved to each company. In comparison to this, there were only three lonely doctors who came on three separate occasions to protect the interest of the medical profession, wherein, many millions of dollars of the doctors' money, and human lives were at stake.

Members of this Association, we cannot continue to go on in this languid and careless fashion, expecting to place the high calling of our profession on a sound business basis. I am not advocating that the doctor or this medical association forestall any of its scientific advancements to further the political ambitions of any individual, but I am calling to your mind, that science and business in our profession must go hand in hand. That we cannot expect a farmer, lawyer or a banker to make laws that are just and equitable to the medical profession. The medical profession themselves must have a representation in the law making body. The doctors should exert their influence upon the members of the legislature. In other words, I call upon the medical men within the hearing of my voice to take an active part in the election of public officials, especially to lend their influence to the election of state officials and to become intimately acquainted with their representatives and senators.

I will also suggest that the Oklahoma State

Look at This Fracture Suspension Frame



A sample will be sent to any reliable surgeon or Hospital in order that they may try it out. It has the following features as to fractures:

1. Fracture of the lower extremities.
2. Diabetic ulcers, elevation and heat treatment.
3. Arthritic cases, elevation and heat treatment.
4. Buerger's Disease, elevation and heat treatment.
5. To prevent shortening deformity after fracture.
6. Various forms of paralyses where heat and elevation are indicated, and varicose ulcers.
7. It eliminates cumbersome scaffolding around bed.
8. It eliminates plaster casts.
9. It has five point adjustable suspension, longitudinal and transverse.
10. It is made of aluminum, does not show in X-ray, is light in weight and is non-corosive.
11. It has the proper knee angle as approved by the Medical profession, affording two ways preventing anklylosis.

It is not only endorsed but in daily use by Oklahoma Orthopedists and surgeons.

We would like for you to place your order for one of these suspension frames at the reduced price of only **$35.00—$10.00 cash, and $5.00 per month on balance.**

G. LANGE

Muskogee Artificial Limb and Appliance Company

MUSKOGEE, OKLAHOMA

Medical Association at this session appoint a special committee; to consider the present public health laws of the state, to outline a complete program for the care of the sick so that the burden can be equally borne by all; and to advance measures to enact as laws that will aid the doctor to collect his just fees. This committee report can be studied and ratified by the association at its next annual meeting after which a uniform influence should be brought to bear on the legislature which will meet in 1935, so that the entire program of medical advancement and equality can be made a law.

LOUIS H. RITZHAUPT, M.D.

REPORT OF EXAMINATION FOR LICENSES TO PRACTICE MEDICINE

Examination held at State Capitol, Oklahoma City, June 7th and 8th, 1933. The following applicants passed:

Name	Year of Birth	Place of Birth	School of Graduation	Year of Graduation	Home Address or Previous Location
Evans, Fred Green	1903	Tiptonville, Tenn.	Univ. of Tenn.	1932	Afton, Okla.
Loney, Wm. Robt. Roy	1901	Hope, N. D.	Univ. of Minn.	1926	Tulsa
Russell, Geo. Richard	1899	Hamler, Ohio	Western Reserve	1899	Tulsa
Beatty, Samuel Richard	1903	Beverly, Ohio	Univ. of Wis.	1932	Okla. City
Mattison, Wm. Lawrence	1903	Columbia, S. C.	Northwestern	1932	Calvin Okla.
Sewell, Dan Roy Jr.		Jacksonboro, Tex	Wash. Univ.	1931	Okla. City
Serwer, Milton John	1904	Detroit, Mich.	Rush Med. Col.	1931	Okla. City
Peacock, Berenice Grace	1900	San Francisco, Calif.	Univ. of Calif.	1928	Okla. City
Reed, Charles Bruce	1909	Denison, Tex.	Baylor Med. Col.	1932	Durant, Okla.

(The following took the examination, and passed, and licenses will be issued when approved internship certificate is filed with the Secretary).

Winston, John R.	1908	McMillan, Okla.	Univ. of Okla.	1933	McMillan, Okla.
Fryer, Samuel R.	1910	Muskogee, Okla.	Univ. of Okla.	1933	Okla. City
Traverse, Clifford A.	1908	Byron, Okla.	Univ. of Okla.	1933	Okla. City
Wainwright, Tom Lyon	1909	Hattiesbur, Miss.	Univ. of Okla.	1933	Okla. City
Reed, Karl Asbury	1902	Guthrie, Okla.	Univ. of Okla.	1933	Okla. City
Tisdal, William C.	1907	Cordell, Okla.	Univ. of Okla.	1933	Elk City, Okla.
Angus, Donald A.	1907	Lawton, Okla	Univ. of Okla.	1933	Okla. City
Ray, Raymond G.	1907	Downs, Kan.	Univ. of Okla.	1933	Okla. City
Barb, Thomas J.	1910	Merkel, Tex.	Univ. of Okla.	1933	Norman, Okla.
Shelby, Hudson S.	——	Savannah, Tenn.	Univ. of Okla.	1933	Okla. City
Luton, James P.	1907	Lindsay, Okla.	Univ. of Okla.	1933	Okla. City
Russell, Lum E.	1910	Cyril, Okla.	Univ. of Okla.	1933	Okla. City
Coppedge, Orville N.	1902	Jennings, Okla.	Univ. of Okla.	1933	Okla. City
McMillan, James M.	1904	Atlee, Ark.	Univ. of Okla.	1933	Okla. City
Hackler, John F.	1910	Tahlequah, Okla.	Univ. of Okla.	1933	Tahlequah, Okla.
Crane, Francis S.	1909	Texas	Univ. of Okla.	1933	Chandler, Okla.
Smithson, Carl B.	1906	Alva, Okla.	Univ. of Okla.	1933	Okla. City
Sturm, Chas. Edward	1905	Marysville, Mo.	Creighton Med. Col.	1933	Okla. City
Eads, Chas. H.	1903	Mt. Carbon, W. Va.	Univ. of Okla.	1933	Tulsa
King, Everett G.	1910	Colony, Okla.	Univ. of Okla.	1933	Cordell, Okla.
Tichenor, Ernest L.	1903	Morganfield, Ky.	Univ. of Okla.	1933	Okla. City
Breco, Davis	1899	Canton, Tex.	Univ. of Okla.	1933	Ada, Okla.
Haynie, Weldon K.	1907	Aylesworth, Okla	Univ. of Okla.	1933	Durant, Okla.
Greer, Rex	1906	Woodford, Okla.	Univ. of Okla.	1933	Okla. City
Huggins, James R.	1904	Chickasha, Okla.	Univ. of Okla.	1933	Okla. City
Ruth, Weldon K.	1909	Okeene, Okla.	Univ. of Okla.	1933	Okeene, Okla.
Reed, Chas. W.	1909	Okla.	Univ. of Okla.	1933	Okla. City
Messenbaugh, Joseph F.	1910	Okla. City	Univ. of Okla.	1933	Okla. City
Perry, Fred T.	1908	Texas	Univ. of Okla.	1933	Okla. City
Ford, Harry C.	1908	Middletown, Mo.	Univ. of Okla.	1933	Tulsa
Woodson, Orville M.	1907	Poteau, Okla.	Univ. of Okla.	1933	Poteau, Okla.
Smith, Wendell L.	1907	Topeka, Kan.	Univ. of Okla.	1933	Tulsa
Lingenfelter, Paul B.	1907	Clinton, Okla.	Univ. of Okla.	1933	Okla. City
Shipp, Jesse D.	1910	Hugo, Okla.	Univ. of Okla.	1933	Idabel, Okla.
Bailey, Carl H.	1909	Ft. Worth, Tex.	Univ. of Okla.	1933	Okla. City
Peter, Maurice L.	——	Oxford, Kan.	Univ. of Okla.	1933	Okla. City
MacLeod, Sherburne	1906	May, Okla.	Univ. of Okla.	1933	Hooker, Okla.
Butts, Imogene	1909	Holdenville, Ok.	Univ. of Okla.	1933	Holdenville, Okla.
Sturgeon, H. Violet	1908	Hennessey, Okla.	Univ. of Okla.	1933	Okla. City
Cantrell, Emma Jean	1907	Mounds, Okla.	Univ. of Okla.	1933	Okla. City
Craft, Wilma	1908	Vermont, Ill.	Univ. of Okla.	1933	Okla. City
Funk, Gustavus DeLana	1904	Marysville, Mo.	Univ. of Okla.	1933	El Reno, Okla.
Watson, Price T.	——	Erick, Okla.	Univ. of Okla.	1933	Blair, Okla.
Jobe, Virgil Ro.	1902	Lincoln, Ark.	Univ. of Okla.	1933	Okla. City
Hoover, Wilkie Dee	1908	Wynnewood, Ok.	Univ. of Okla.	1933	Tulsa
Darrough, James B.	1910	Vinita, Okla.	Univ. of Okla.	1933	Vinita, Okla.
Lambke, Phil M.	——	Byron, Okla.	Univ. of Okla.	1933	Amorita, Okla.
Speed, Henry K. Jr.	1909	Sayre, Okla.	Univ. of Okla.	1933	Sayre, Okla.
Hackler, Harold W.	1906	Westville, Okla.	Univ. of Okla.	1933	Tahlequah, Okla.
Kerr, Walter C. H.	1903	Peoria, Ill.	Univ. of Okla.	1933	Clinton, Okla.
Parsons, Orval L.	1907	Enid, Okla.	Univ. of Okla.	1933	Okla. City
House, Rex Clayton	1909	Carnegie, Okla.	Univ. of Okla.	1933	Okla. City
Shiflet, Albert Woods	1906	Lebanon, Mo.	Univ. of Okla.	1933	Okla. City
Rogers, Galen A.	1906	Waynoka, Okla.	Univ. of Okla.	1933	Nicoma Park, Ok.
Curb, Delos Griffin	1897	Leon, Okla.	Univ. of Okla.	1933	Okla. City
Lee, Robert Ray	——	Dublin, Tex.	Univ. of Okla.	1933	Okla. City